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THE ASSOCIATION OF JAUNDICE AND ENLARGED SPLEEN, AND SPLENOMEGALIC JAUNDICE.*

By H. D. ROLLESTON, M.D., F.R.C.P.,
Senior Physician to St. George's Hospital; Physician, Victoria Hospital for Children.

SPLENOMEGALIC jaundice simply means that jaundice is associated with splenic enlargement, but this descriptive title is only employed when the association is not satisfactorily explained by some well-recognised cause, such as hypertrophic biliary cirrhosis or the presence of undoubted syphilis. The combination of splenomegaly and jaundice does not of course constitute a definite disease, and the cases which, since they present these two symptoms as the main or only morbid changes, come under this heading, may be due to distinct causes.

It is rather remarkable that the combination of jaundice and splenic enlargement is not more commonly seen as the result of mechanical pressure exerted in the portal fissure simultaneously on the portal vein (causing splenomegaly) and on the bile-duct (causing jaundice). Although backward pressure, as in heart disease, with nutmeg liver, does not produce splenic enlargement, thrombosis of the portal vein, implicating the splenic vein, causes very notable enlargement of the spleen. As a matter of experience, however, it is extremely rare to find considerable splenic enlargement in cases of jaundice due to malignant disease in the portal fissure.

The combination of considerable splenic enlargement and jaundice may be conveniently considered under two headings: (A) as occurring in well-recognised diseases, and (B) when these two signs constitute the main or only clinical manifestations, or splenomegalic jaundice.

* A Lecture given at the Polyclinic.

(A) Splenomegaly and Jaundice in the Course of Recognised Diseases.

It is unnecessary and impossible to give a detailed description of the various conditions in which splenic enlargement and jaundice may be combined. In considering this subject it will be convenient first to enumerate the conditions in which enlargement of the liver may be combined with splenomegaly and jaundice.

(*a*) In ordinary cases of portal cirrhosis of the liver the spleen, though usually enlarged, is not so to any considerable extent, and jaundice occurs only as a temporary and almost accidental complication in the course of the disease. Still, every now and then we meet with cases of portal cirrhosis presenting considerable splenic enlargement and jaundice. This is particularly likely to occur in cases of cirrhosis which show a transition from ordinary (portal, multilobular) cirrhosis to the hypertrophic biliary cirrhosis. In some of these cases the cirrhosis is advancing rapidly so that they are relatively acute, and the patients are commonly in a toxæmic condition, and rapidly going down hill. They are often extremely alcoholic, and this, by setting up gastro-duodenitis and catarrhal changes in the bile-duct and head of the pancreas, may account for the jaundice. On the other hand the "mixed" form of cirrhosis found in these cases after death may include changes in the intra-hepatic ducts which would account for some degree of icterus. In rare instances, ordinary cirrhosis accompanied by the usual amount of splenic enlargement may be complicated by the presence of a gall-stone in the common bile-duct, and therefore by jaundice.

(*b*) *Hypertrophic biliary cirrhosis* (*Hanot's cirrhosis*).—There has been some difference of opinion on the question whether this clinical form of cirrhosis is sufficiently distinct from the varieties of ordinary cirrhosis to justify its separate consideration. Quite recently Oertel,* from a careful study of the morbid appearances, concludes that a separation between the two cannot be upheld. There is, however, a good deal to be said in favour of recognising the condition, which is, it must be admitted, rare, and certainly shows transitional forms to ordinary cirrhosis. It appears to me that the state of affairs is much the same as it is in regard to the large white kidney (chronic tubal, or parenchymatous nephritis), and the contracted white kidney, which we may consider as corresponding to hypertrophic biliary and to portal cirrhosis respectively. The two forms of renal disease are now regarded as distinct, though some writers have considered that the large white becomes the small contracted white kidney in course of time. My own opinion is that hypertrophic biliary cirrhosis should be recognised as a disease with the following distinctive characters: chronic jaundice of varying intensity but never completely obstructive; enlargement of the liver, and, as compared with portal cirrhosis, more splenomegaly; absence of ascites and hæmatemesis; incidence in early adult life, and not specially related to alcoholism. Structurally, the cirrhosis is primarily monolobular in type.

(*c*) In congenital obliteration of the bile-ducts the liver and spleen are both enlarged, and there is deep jaundice. This condition is seen very early in life, and practically all the cases die within eight months of birth; in sixty-two cases collected by Lavenson* three only survived eight months, one to eleven months.

(*d*) By Banti's disease is meant cirrhosis of the liver with jaundice, as the result of chronic splenic anæmia of adults. Sometimes the title "Banti's disease" is applied to cases of chronic splenic anæmia before the hepatic fibrosis and jaundice have supervened, but it is most convenient to employ the term as stated above. It has been thought that toxins manufactured in the spleen are conveyed by the splenic and portal veins to the liver and in course of time set up cirrhosis (Chauffard); in other words, that portal cirrhosis of the liver may be due to poisons conveyed by either of the two main tributaries of the portal vein, the splenic in this instance and the mesenteric veins in ordinary cases. Thus, as the result of a long-continued toxic splenitis (chronic splenic anæmia) cirrhosis of the liver may result.

The existence of Banti's disease, and also of chronic splenic anæmia, has been much questioned, especially in France, and it has been thought to be the result of latent cirrhosis, to be due to backward pressure of heart failure, or to be a remote effect of syphilis. Some cases clinically characteristic of chronic splenic anæmia and of Banti's disease

* Oertel, 'Arch. Int. Med.,' Chicago, 1908, i, 394.

* Lavenson, 'Journ. Med. Res.,' Boston, 1908, xviii, 61.

have undoubtedly turned out to be due to syphilis, but this explanation certainly will not fit all the cases.

(*e*) New growths of the liver often give rise to jaundice, but very seldom induce enlargement of the spleen. In rare instances lymphadenoma may give rise to hepatic and splenic enlargement, and, by pressure on the common bile-duct, to jaundice; in cases in which the change is mainly in the abdominal glands and viscera the diagnosis may be extremely difficult.

(*f*) *Syphilis.*—Among the recognised diseases that may give rise to splenomegaly and jaundice, probably the most difficult to assess at its true importance is syphilis. For cases in which there is nothing to suggest syphilis may recover on anti-syphilitic treatment, or after death show unmistakable signs of lues veneris. It should also be remembered that in syphilitic disease of the liver and spleen there may, just as in cerebral syphilis, be no outward sign of the disease. Another reason why it is so difficult to appraise the causal importance of syphilis in this connection is that, although syphilis does not very commonly induce jaundice, it may do so in a number of different ways. Thus, in inherited syphilis jaundice is rare; it may be due to the changes in the liver which involve the small bile-ducts, it may depend on secondary infection with *B. coli*, *B. proteus*; or in very rare instances it may be the result of syphilitic disease of the common bile-duct, as in a case I recorded in 1907,* in which clinically the features were those of congenital obliteration of the bile ducts. In inherited syphilis the spleen is enlarged in about half the cases, so that when jaundice does occur it is not uncommon to get the two associated. I have watched a child with an enormous spleen reaching down to the iliac crests, slight jaundice, and at first intense toxæmia, gradually recover under good hygienic conditions and mercurials, which were given, not because there was any definite evidence of syphilis, but rather on the chance that it might have some good effect; after two years' time the spleen could not be felt.

In "tardive" or delayed hereditary syphilis, in which symptoms resembling those of the tertiary stage come on in adolescence or early adult life, jaundice is not often seen, but occasionally cases with great hepatic and splenic enlargement and jaundice occur. In cases of delayed hereditary syphilis the splenic enlargement is commonly due to lardaceous disease, but a boy, æt. 17 years, under the care of my colleague Dr. Ewart, presented extreme splenic enlargement and chronic jaundice with gummata on the long bones. The post-mortem examination showed that there was not any lardaceous change in the spleen. A case of this kind suggests that the splenic enlargement, though undoubtedly occurring in the subject of delayed inherited syphilis, might be due to some secondary infection or morbid process which had been able to take root in tissues devitalised by the effects of syphilis.

In the early stages of acquired syphilis some slight degree of splenic enlargement is common, and in rather rare cases, it is said in about 2 per cent. of all cases of syphilis, jaundice supervenes, often synchronously with the roseola, so that the combination of splenomegaly and jaundice results. This form of jaundice is interesting from several points of view; first, it is specific, and yields to mercurial treatment and not to the usual remedies employed for ordinary benign jaundice; secondly, it is more often seen in women than in men; and thirdly, it may pass into acute yellow atrophy. In fifty collected cases of post-syphilitic acute yellow atrophy there was again a great preponderance of females, only ten being males (Fischer).

In the tertiary stage of acquired syphilis jaundice, though far from common, may be due to pressure on the bile-ducts in almost any part of their course by gummata or cicatrices, and as the spleen may be enlarged from lardaceous change, fibrosis, or in rare instances gumma, the association of splenomegaly and jaundice may be met with as a late result of syphilis.

It will now be convenient to mention some other recognised diseases in which jaundice and enlarged spleen may concur, but in which, from the relative insignificance of these features and from the prominence of other symptoms, there is no doubt as to the primary condition.

In acute yellow atrophy there may be some splenic enlargement, but it is never a very notable feature. In Blackwater fever the two conditions are present. In pernicious anæmia neither jaundice nor clinical enlargement of the spleen is often seen. Occasionally, however, the lemon or primrose colour of the skin is exaggerated, and Chauffard has recently divided cases of pernicious anæmia

* Rolleston, 'Brit. Med. Journ.,' 1907, vol. ii, p. 947.

into those without and those with jaundice. In rare cases of wandering spleen the traction exerted on the duodenum has been known to give rise to jaundice (Treves). In tuberculosis of the liver jaundice practically never occurs, but occasionally tuberculous glands in the portal fissure may compress the bile-duct and so induce jaundice. In some cases there is considerable enlargement of the spleen from tuberculosis; in rare instances the spleen shows so much more tuberculosis than any other part of the body that the condition is spoken of as primary massive tuberculosis of the spleen; it is conceivable that in these rare cases concomitant tuberculosis of the lymphatic glands in the portal fissure might induce jaundice and so give rise to the combination of jaundice and splenic enlargement.

(B) Splenomegalic Jaundice.

As already pointed out, this title is conferred on those cases in which no underlying cause or other recognised disease can be made out. These cases of considerable splenomegaly and very chronic jaundice of varying degree are extremely remarkable; they are probably due to different causes, but our knowledge on this subject is very scanty, and there are accordingly a number of speculations and hypotheses as to their explanation.

It is difficult to know how best to consider the subject, but perhaps it will be most convenient to divide the cases into groups, which, however, are far from being absolutely distinct, or, so to speak, water-tight. The groups are composed of cases which are (1) hereditary, (2) congenital, and (3) acquired.

(1) *Hereditary splenomegalic jaundice.*—A number of cases have been recorded in which enlarged spleen and chronic jaundice are hereditary. Sometimes the condition is also congenital, sometimes it comes on years after birth; and the same family may show cases of splenomegalic jaundice coming on after birth in one generation, and in the next generation cases of congenital jaundice and splenomegaly. Thus, Benjamin and Sluka* record jaundice in grandfather, father, and son; in the two later generations jaundice had existed from birth, and was associated with splenomegaly. This suggests that the morbid process becomes intensified in transmission.

The interesting way in which the disease may vary in transmission is shown by the history of a case related by Hayem; the maternal grandfather had ulcers on the legs and jaundice from the age of twenty; the mother was jaundiced from birth, and had attacks of abdominal colic, and her son, the patient, æt. 39 years, became jaundiced at the age of seventeen; in this series the intensity of the process showed some signs of abating.

In hereditary jaundice there are notable variations in its transmission and in its degree; one only of several brothers and sisters may be affected, one only may show chronic jaundice whilst others may have had passing attacks of jaundice, thus showing the family tendency. This family tendency to jaundice of varying degrees has been definitely crystallised out by Gilbert and Lereboullet in their numerous contributions on family cholæmia. This is not a disease but rather a diathesis, in virtue of which the biliary system is a place of diminished resistance, and therefore prone to become infected and inflamed on slight provocation. The subjects of it—often Eastern in origin or Jews—are dark, bilious-looking but not definitely jaundiced, and the blood serum contains bile-pigment, but there is no bile in the urine. They are prone to various manifestations, such as dyspepsia, melancholy, slow pulse, pigmentation of the skin, xanthelasma, and are apt to develop transient or chronic jaundice and cirrhosis of the liver. Just as some families are prone to emphysema and bronchitis, so others are specially liable to attenuated biliary infections.

Although cases of hereditary splenomegalic jaundice are distinct from Hanot's hypertrophic biliary cirrhosis, inasmuch as the liver is not enlarged, it must be at once admitted that they are related and both form links in the chain of closely-related diseases, comprising chronic splenic anæmia, splenomegalic jaundice (hereditary and congenital forms), Banti's disease, meta-splenomegalic biliary cirrhosis in which the splenic enlargement precedes any manifest change in the liver, and hypertrophic biliary cirrhosis. This close association between hereditary splenomegalic jaundice and Hanot's cirrhosis is shown by two cases of jaundice since birth occurring in father and son; the father had enlargement of both spleen and liver, the son of the spleen only (Lortat-Jacob and Sabaréanu).* The

* Benjamin und Sluka, 'Berlin. klin. Wochens.,' 1907, xliv, 1065.

* Lortat-Jacob et Sabaréanu, 'Rev. de méd.,' Paris, 1904, xxiv, 810.

father's condition would very closely resemble Hanot's disease.

(2) *Chronic congenital splenomegalic jaundice.*—This may be hereditary or may be met with in individuals without any family history of jaundice. The hereditary cases have just been referred to, and it has been shown that these two groups are closely allied. There is probably no very good ground for dividing the hereditary from the isolated cases of persistent congenital jaundice, for it is conceivable that the isolated cases are the first examples of an hereditary series.

As in the hereditary but not congenital cases, the disease may become intensified in the second and subsequent generations. Thus, in two cases of congenital hæmolytic jaundice in father and daughter, the daughter showed more anæmia and jaundice (Widal, Abrami, et Brulé).*

Pathogeny of the hereditary and congenital forms.—Although the explanation of those cases of chronic splenomegalic jaundice may not be the same, it is more convenient to attempt discussion of this difficult subject under one heading than to do so separately because (1) these forms have some manifest symptoms in common, namely, varying anæmia, chronic jaundice, splenomegaly; (2) hereditary jaundice may be congenital.

Several hypotheses have been put forward to explain the condition. For the congenital cases it has naturally been suggested that jaundice is due to some malformation of the bile-ducts. In some cases this has been definitely disproved; and as there is no evidence in favour of this *à priori* assumption, it need not be further alluded to. The same applies to the suggestion that the jaundice is the result of an abnormal communication between the bile-ducts and the lymphatics which allows the bile to pass into the circulation. Hayem has lately stated his belief that the cases are due to syphilis; though this may be true in isolated examples there is no proof that it explains the majority, much less all of the cases.

The two hypotheses to which we must devote a few words are (*a*) that the condition is due to cholangitis, and (*b*) that it is due to active hæmolysis in the spleen.

(*a*) The cholangitic hypothesis is to the effect that the jaundice is due to the inflammation of the ducts in the liver which produces obstruction. This cholangitis must be due to some infection or to a poison, which is probably excreted from the blood into the ducts and thus sets up catarrh. The poison might be manufactured in the alimentary canal, in cases of hereditary and congenital jaundice possibly as the result of some as yet unknown freak of metabolism analogous to the chemical malformations (as they have been called by Garrod), which give rise to cystinuria and alcaptonuria. In hereditary cases in which the jaundice does not date from birth it is possible that owing to some want of vital resistance the intestines become the site of some special form of infection or auto-intoxication, and that the poisons thus formed set up catarrhal cholangitis.

Although the alimentary canal is the most obvious source of infection or intoxication it is possible that the infection or toxic bodies responsible for splenomegalic jaundice may be derived from other parts of the body. It is not necessary to assume that the condition is the same in every instance; it is quite possible that in some instances it is derived from the alimentary canal, and in others from other parts of the body. Thus, in favour of the view that the original focus of intoxication or infection may be in the general venous systemic system, it may be mentioned that in a few cases of hereditary chronic splenomegalic jaundice there have been chronic ulcers on the legs or elsewhere on the body (Barlow and Shaw,* Hayem). This, however, is so infrequently mentioned that it would appear probable that in most instances the primary site of absorption is in the alimentary canal.

Cases of congenital and hereditary splenomegalic jaundice usually show anæmia, and it must be supposed that the same poison which sets up cholangitis is responsible for the anæmia. The anæmia might be hæmolytic and due to the destructive action of the poison on the red blood-corpuscles; this seems probable on the analogy of toluylenediamine, which gives rise both to hæmolytic anæmia and to jaundice. It is, however, conceivable that the icterogenetic poison might inhibit the production of red blood-corpuscles by the red marrow —a kind of aplastic anæmia.

The splenic enlargement has been explained in

* Widal, Abrami, et Brulé, 'Compt. rend. Soc. biol.,' Paris, 1908, lxiv, 655.

* Barlow and Shaw, 'Trans. Clin. Soc.,' London, 1902, xxxv, 155.

various ways; the most obvious view is that the same infection or intoxication which leads to cholangitis and anæmia sets up proliferative changes in the spleen. Thus, in 1898, Hayem * described chronic splenomegalic infective jaundice, which he now considers to be due to inherited syphilis.

The other suggestions appear improbable; it has been asserted that the splenomegaly in these cases is a compensatory process to antagonise the hæmolysing effect of the bile acids, which should, as the result of biliary obstruction, pass into the blood (Rist and Ribadeau-Dumas †); and it has been stated that in obstructive jaundice the blood serum acquires anti-hæmolytic properties, that the resistance of the red blood-corpuscles to hæmolytic agents is increased, and that they become larger in size. The suggestion that the spleen antagonises the hæmolytic action of bile acids, and therefore becomes enlarged, can hardly be entertained. Jaundice due to obstruction of the larger bile-ducts, for example by the pressure exerted by tumours, is accompanied by more bile acids in the urine, and so presumably in the blood, than is the jaundice due to obstruction of the small intra-hepatic bile-ducts, and yet enlargement of the spleen is practically never seen in uncomplicated cases of mechanical obstruction of the larger ducts. Again, there are some cases of congenital jaundice which persist throughout life but show no enlargement of the spleen (Cocking,‡ Pitt,§ Glaister ||).

Another suggestion to explain splenomegaly in jaundice due to chronic intra-hepatic cholangitis is that there is some peri-cholangitic fibrosis, and that this gives rise to portal obstruction, backward pressure, and thereby splenic enlargement (Lereboullet ¶). This explanation seems to be extremely unlikely, for although the spleen is enlarged in ordinary cirrhosis the splenomegaly is not nearly so great as in these cases of peri-cholangitis, in which the fibrosis is incomparably less than in ordinary portal cirrhosis. Again, in some cases of peri-cholangitic fibrosis such as occasionally occur in cholelithiasis, the spleen is little if at all enlarged.

(*b*) *Hypothesis that splenomegalic jaundice is due to active destruction of blood in the spleen.*—According to this conception there is no infection of the bile-ducts or cholangitis to cause the jaundice, which is accounted for in a not very convincing manner by the supposition that the large amount of pigment present thickens the bile and so obstructs the flow. This conception has recently been put forward by Chauffard and his pupils, and supported by a considerable amount of work on the examination of the blood, so that it is necessary to refer to it although at first sight it may seem improbable. This hypothesis was originated in 1900 by Minkowski, who described a case of chronic hereditary jaundice with enlarged spleen, urobilinuria, and renal fibrosis in a man æt. 42 years, dead from pneumonia. At the autopsy there was no definite disease of the liver, and the bile-ducts were healthy except for a pigment calculus in the gall-bladder. Minkowski considered that there was a primary lesion of the spleen.

Chauffard,* in 1907 and 1908, elaborated this view that the congenital splenomegalic jaundice seen in patients surviving into adult life depends on an active hæmolytic process in the spleen. The histological evidence in favour of this is rather slender, and appears to be based on one case showing intense congestion and macrophagic reaction in the spleen (Vaquez and Giroux).

In these cases of congenital jaundice in adults the blood is described as differing markedly from cases of ordinary jaundice. These particular changes in the blood have attracted little or no attention in this country, and certainly require critical examination and confirmation. In the congenital (hæmolytic) jaundice of adults the red blood-corpuscles are described as more fragile or breaking down much more readily than in health. This hæmolytic process was not due to the blood serum but presumably to the local hæmolytic activity of the spleen; the corpuscles are smaller than normal (microcythæmia), and show granular degeneration, as in grave anæmias particularly that of lead poisoning. On the other hand, in ordinary obstructive jaundice the converse conditions are described, namely, increased resistance of the red blood-corpuscles to hæmolysis and increased size

* Hayem, 'Presse méd.,' Paris, 1898, i, 121; 'Bull. et mém. soc. méd. des hôp. de Paris.,' 1908, xxv, 122.

† Rist et Ribadeau, 'Tribune méd.,' Paris, 1906.

‡ Cocking, 'Quart. med. Journ.,' (Sheffield), 1903, xi, 104.

§ Pick, 'Wien. klin. Wochenschr.,' 1903, xvi, 493.

|| Glaister, 'Lancet,' 1879, i, 295.

¶ Lereboullet, 'Semaine méd.,' Paris, 1903, xxiii, 180.

* Chauffard, "Pathogénie de l'ictère congénital de l'adulte," 'Semaine méd.,' Paris, 1907, xxvii; 1908, xxviii, 48.

in the individual erythrocytes. Chauffard thus argues in favour of the conclusion that some cases of chronic splenic enlargement and jaundice are due to active hæmolysis in the spleen.

It has been urged that this discussion is not merely of academic and pathological interest, but that it is of practical importance, for if the cause of the jaundice is cholangitis it would be amenable to surgical treatment, namely, drainage of the larger ducts, whereas in the cases of purely hæmolytic jaundice no benefit can be expected from this procedure.

Until further information is available we must keep an open mind, but at present the conception of chronic jaundice due to a primary hæmolysing splenomegaly is very far from proved. Hæmolytic jaundice, due to poisons which set up cholangitis, and at the same time induce hæmolytic anæmia, is well established, as shown by Stadelmann's and Hunter's experiments with toluylenediamine. It therefore seems more probable that chronic splenomegalic jaundice is the result of an analogous but chronic toxæmia, than that it is due to excessive hæmolytic activity of the spleen, especially as it is not clear what causes this excessive hæmolytic activity.

(3) *Acquired splenomegalic jaundice.*—Isolated cases of jaundice with splenic enlargement, but without any other obvious manifestation except a varying degree of anæmia, come under observation from time to time. From this category it is convenient to exclude cases of jaundice which are hereditary. It cannot be said that these cases constitute a definite disease, as the symptoms may be due to various causes.

In both children and adults acquired splenomegalic jaundice may be only an early stage of Hanot's hypertrophic cirrhosis, or may in reality be an irregular manifestation of syphilitic infection. Although we must guard against referring unusual conditions to syphilis without some fairly good reason, it may be well to try the therapeutic test in doubtful cases. Bernard* records the case of a man who, some years after contracting syphilis, became jaundiced, and was found to have an enlarged spleen; both of these manifestations disappeared after intra-muscular injections of benzoate of mercury. Subsequently these symptoms returned and were again removed by a repetition of the mercurial treatment.

As a rule the cases of acquired splenomegalic jaundice suffer more from their condition than do cases of chronic congenital splenomegalic jaundice. It may very reasonably be argued that if the patient is able to survive jaundice dating from birth, some degree of immunity has been evolved, and that the condition is not extremely serious. Chauffard has insisted that the acquired cases of splenomegalic jaundice show more anæmia and less jaundice as compared with congenital cases.

Jaundice is usually somewhat slight, but both it and the condition of the blood vary in degree from time to time, the patient being worse when it becomes deeper, thus resembling pernicious anæmia. In the intervals the jaundice may so far disappear that the case then presents the features of chronic splenic anæmia, and thus shows the close relationship that exists between these diseases; in fact these cases might be described as splenomegaly with intermittent jaundice.

The *clinical characters* of the different forms of splenomegalic jaundice vary, as has been incidentally pointed out; thus the acquired cases suffer more and are often ill. The jaundice is not complete, except possibly during acute exacerbations, and is often comparatively slight; in such cases there may be no bile in the urine for considerable periods (acholuric jaundice). The chronic congenital cases, on the other hand, are often remarkably free from symptoms, and this is in marked contrast to the very noticeable jaundice and splenomegaly. In fact, in the chronic congenital cases it does not necessarily interfere with an active and prolonged life. In some instances, however, exacerbations occur, as if some infection attacked the spleen, which is of course a place of diminished resistance; the spleen may then increase in size and the liver enlarge, thus suggesting that the condition is one of biliary cirrhosis, into which some cases at first regarded as splenomegalic jaundice may eventually pass. In some cases there is interference with growth and development, so that the patients look much less than their years. The fingers may be clubbed as in hypertrophic biliary cirrhosis.

The treatment of cases of splenomegalic jaundice consists (1) in eliminating the factor of syphilitic infection, and (2) in putting the patient under the best hygienic conditions—fresh air, sun, and dry soil, if necessary a change of residence, for example, from a damp depressing district.

(3) As the condition may depend on cholangitis due to intestinal infection and toxæmia, the diet should be simple and free from irritating, spicy constituents. Guaiacol in five-grain doses, twice or three times a day with food, should be given a good trial to minimise intestinal disturbance. Salicylate of sodium, gr. x, combined with urotropine, gr. v, may also be given twice or three times daily; urotropine exerts an antiseptic action on the biliary tract and also on the intestine, and salicylate of sodium increases the flow of bile and so washes out the ducts.

October 12th, 1908.

* Bernard, L., 'Bull. et mém. soc. méd. des hôp. de Paris,' 1908, xxv, 158.

ON THE MODERN TREATMENT OF FRACTURES BY MEANS OF DIRECT INTERNAL SPLINTAGE, WITH A NOTE ON THE TREATMENT OF FRACTURES OF THE SHAFTS OF THE FEMUR AND TIBIA.*

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THE treatment of fractures has always been a department of surgery encompassed with many difficulties—the despair and perhaps the scandal of many practitioners. Of recent years in particular the use of skiagraphs has laid bare before the people in general and ourselves in particular the fact that under the present methods of treatment a broken bone is rarely, if ever, restored to its original shape. Perhaps a skiagraph taken some time after the fracture is shown to a jury and the medical man mulcted of damages; it may be for no fault of his own. The public have a right to expect a fairly correct diagnosis in a case of fracture, and the use of the X rays is most helpful in arriving at such a diagnosis. It is, therefore, foolish to neglect such help whenever it can be obtained. But a man who breaks a bone has no right to expect a perfect result and to complain if he does not regain every ability which he possessed prior to the injury. If he smashes his motor car, it may be, and usually is, impossible to repair it so finely that the engine, with no new parts, is as good as if no accident had happened to it. Hence, it is unjust to expect perfection in all cases of the treatment of fractures. Let us then examine the general features of the methods of treatment at our disposal. They consist of splints, massage, or both. The treatment by massage is the more modern, and is a most useful adjunct. But the treatment by splintage is a very old method. In fact it originated at the very dawn of surgery. The ancient method of the treatment of fractures by splintage has not progressed to anything like the extent which other and more recent branches of surgery have, such as the treatment in abdominal surgery. The subject of this lecture is to put the question of the modern treatment of fractures before you.

First of all, what are the general features of this treatment by splintage? Splints are applied to the outside of the injured part in order to keep it at rest, to hold the fragments of the broken bone together and in the proper position in which they are to unite. Splints easily accomplish the first condition—keeping the part at rest—but fulfil the other two conditions imperfectly, because they are placed along the outside of the limb and are separated from the bones they are to hold in apposition by skin, fat, muscle, extravasated blood, etc.; and, in addition, they cannot be applied tightly without interfering with the blood supply of the part, perhaps cutting it off altogether and causing gangrene. In consequence, they have to be bandaged and fixed with tightness short of interfering with the blood supply, with the result that the fragments of the broken bone can move upon each other within the splints. The force of this argument will be appreciated if an attempt is made to fix in good apposition two portions of a broken walking stick inside a pillow by means of splints bound lightly along the outside. The attempt is childish. Splints which are placed along the outside of the limb may be termed *external splints*. And the method, as the splints are not placed directly on the fragments, may be called *indirect splintage*. On the whole the clinical results of this rough and inexact method are good. But, like everything else in this world, the method has its limitations, so that it must not be expected to do everything. Considering the ancient origin and lineage of this method of indirect treatment of fractures by external splints, it is a fair question to ask if modern surgery has devised anything to supplement it. The answer is "Yes"; modern surgery can supplement it by the method of applying "splints" directly to the fragments of the broken bone, the splints being internal and the splintage direct instead of external and indirect. So that the ancient method of treating fractures was by *indirect splintage with external splints* and the modern method is *direct splintage by means of internal splints.*

Naturally internal and direct splintage can only be accomplised by means of an operation. Hence,

* Part of two lectures given at the Polyclinic.

the modern method is a corollary of the advances of modern operative surgery. The internal splints are silver wires, silk, plates, screws, etc., each different fracture requiring different treatment. The operations are often long and difficult, the technical work which the surgeon has to do being often extremely intricate. It will take many years before the treatment of fractures by internal and direct splintage becomes common, or frequently supersedes the more popular and imperfect one of external and indirect splintage. Therefore it will be wise if the modern method be considered merely as supplementary to the more ancient one. And like a good supplement should, it will be of most use at the very places where the older method fails. In such a manner it will be considered.

The method of indirect splintage by means of external splints fails conspicuously in three situations:

(1) In fractures of the shafts of bones, particularly if they are surrounded by thick muscles, as is the femur, or are oblique.

(2) In the neighbourhood of joints, such as the knee.

(3) When one or both of the fragments are too small or too thickly covered for the splints to grasp them, such as is the upper fragment in fractures of the neck of the humerus or femur.

The modern method of direct splintage by means of internal splints will be considered under these headings, but previous to that the limitations and conditions of the method must be stated in order to give a clear idea of its applicability.

(*a*) In the first place internal and direct splintage is the descendant of the modern method of asepsis in the healing of wounds. If a successful result is to be obtained there must be no suppuration, because it would lead to the infection and necrosis of parts of the bone, followed by the separation of sequestra with the local destruction of the part, perhaps of the patient's life as well. When the internal splint is in the neighbourhood of joints there is also to be considered the danger of suppuration in the joint, and, perhaps, the destruction of that joint. A good result can be obtained only by the rigid adherence to the principles of surgical cleanliness. These principles are well known, but there is one which is highly important and perhaps not so well known or appreciated. I have insisted on it elsewhere in an essay upon the "Common Causes of Failure in Surgical Procedures," published in the CLINICAL JOURNAL of September 13th, 1905, pp. 349–352. At an operation the parts surgically most unclean are the patient's, the surgeon's, and assistant's skins. It is impossible in practice humanly to exclude these possible sources of infection. And therein lie the natural limitations of our art. Nothing should ever be put into wounds which has been in contact with any of these dangerous places, such as the handle of the knife. Our ancestors in mediæval times used to employ their fingers when eating the meals. To this day we still use our fingers to eat bread, whilst knives, forks, and spoons have replaced them for meat, soup, and sweets. Indeed, education has advanced so far that it is considered ill-mannered and a sign of bad education to attempt to eat soup or meat with our fingers. A day will surely come when it is regarded as equally bad manners to use fingers at a surgical operation when it can possibly be avoided. Rubber gloves have both a distinct *rôle* and a distinct danger; their *rôle* is to save a surgeon's hands from contamination and from the risk of carrying infection; their *danger* is that they are apt to become a subterfuge for imperfectly washed and surgically unclean hands. In fact, they are subject to as much abuse as chemical antiseptics have been in the "short, perfunctory dip of the hands" in them. To encourage their use in clean cases must be a bad surgical training. The surgery of the future is likely in great part to become what has been called "knife and fork surgery."

(*b*) Thus the first limitation of our modern method is that there must be no suppuration, but the corollary that there must be healing by primary union is less easily attained. This is a curious difference, founded on clinical and not upon bacteriological observations. An operation, say the wiring of some bone, is done; the wound heals except for one place, where a sinus forms, leading to the wire or plate. In due course the foreign body is removed and the wound heals. There is no necrosis of the bone and no obvious question of suppuration. The true explanation lies, I believe, in another direction. Every person, for instance, has a dosage of opium with which his economy can deal and any excess of which will prove fatal; naturally, too, the exact dosage will vary from time to time with his general health. In a similar way every given set of tissues has a certain

dosage of foreign body with which it can deal and encapsulate with fibrous tissue, such as is done to the aseptic ligature after operations. Any excess of this dosage the tissues will be unable to encapsulate, and an "aseptic," or better, non-septic, sinus will form. It is not argued that no micro-organisms are present; the *Staphylococcus albus* probably is present. But it is urged that the prime factor in the non-healing or sinus formation is the over-dosage of the tissues with "foreign body" and not the action of micro-organisms. This explanation was first thrust upon me when using a filigree metal plate to cure a ventral hernia. The presence of the aseptic sinus necessitated eventually the removal of the plate. Hence, failure in primary union must not necessarily be regarded in these cases as due to suppuration, and, therefore, tantamount to the failure of the operation.

(*c*) The use of internal splints has the very great advantage over external splints that massage and passive movements can be begun as soon as the wound has healed or active movements even before that.

(*d*) Internal splints, like external splints, must not be applied too tight. Indeed, Nature makes provision for this. So that if a wire or a screw is used Nature quickly commences to absorb the contiguous hard tissue, loosening them in a few days. After a few weeks they are sometimes quite loose although the operation has been devoid of sepsis. Hence, union between the fragments must begin to take place quickly or the internal splints will become loosened. The internal splintage for broken bones, in which union has been delayed, is only likely to be successful when the delay has been due to some muscle or nerve being interposed between the fragments. Indeed, probably it will be unsuccessful if the delay in the union between the fragments is due to some disease or unfavourable condition of the bones.

(*e*) Internal and direct splintage by operation should be practised in simple fractures if the best results are to be expected from it. Up to recent years cases of compound fracture have been used for it. But compound fractures are apt to be septic, and as asepsis has been urged as a fundamental condition for its success, internal splintage is unlikely to be successful when used in compound fractures. Its merits and demerits must not be judged by the results obtained hitherto in such cases.

It will be seen that the technique of such operations must be very difficult, and will require extreme care and attention for good results to be obtained. Internal splints in no way do away with treatment by external splints—they will only supplement them. Earlier in this lecture it was pointed out that external splints have two chief functions: to put the part at rest, to approximate and appose the fragments. The latter function is much better performed by internal splints, whilst the external splints keep the part at rest and enable the internal splints to do their work.

The femur.—Under this heading will be considered fractures of the shaft of the femur about its middle. The first question to be asked is, "Are the results of treatment by the older period of external splintage satisfactory or not?" The 'St. Thomas's Hospital Reports' are one of the most important sources of information which are available. The 'Reports' are compiled by writers who are absolutely unprejudiced, so that these records are, with their limitations, probably the most truthful and impartial which are extant. During the years 1894 to 1906 the "Surgical Reports" have been compiled by Mr. Cuthbert Wallace, Captain Owen Thurston, Mr. H. J. Marriage, Mr. Edred M. Corner, Mr. P. W. G. Sargent, Mr. C. A. R. Nitch, and Mr. J. E. Adams. During their periods of office as Surgical Registrar 755 cases of fractures of the shaft of the femur were admitted, and of 335 of these, treated by external and indirect splintage, reliable information is given: 21 per cent., or 70, are definitely stated to have had no shortening after treatment; 8 per cent., or 27, ¼ inch or under of shortening; 27 per cent., or 92, ½ inch; 13 per cent., or 42, ¾ inch; 18 per cent., or 59, 1 inch; 2 per cent., or 6, 1¼ inches: 4 per cent., or 15, 1½ inches; 2 per cent., or 8, 1¾ inches; 4 per cent., or 12, 2 inches; 1 per cent., or 4, 2¼ inches.

From these results it will be seen that 21 per cent. had a leg with no shortening and that 81 per cent. had up to one inch of shortening, whilst the mortality was 15 for the 755 cases, just under 2 per cent., the fatal issue being due almost invariably to delirium tremens or some complicating injury. From these figures—87 per cent. with less than an inch of shortening and an all-round mortality of 2 per cent. (in reality unconcerned with the fracture)—it is quite obvious that treatment by

means of internal and direct splintage will have a very uphill task to improve on the old method of external indirect splintage. Still, it is definitely proved that in 13 per cent. of cases of simple fracture of the shaft of the femur the older treatment is unsatisfactory, and it will remain for the more modern treatment to be useful in these cases.

Thus it will be seen that the modern method of the treatment of fractures is not likely to find frequent application in fractures of the femoral shaft.

The tibia.—Fractures of the shaft of the tibia give excellent results when treated by the older method of indirect splintage by means of external splints. But the more oblique the fracture the more easily do the fragments slip on each other and the more difficult are they to hold in position by external splints. These oblique fractures are the result of indirect violence occurring in the lower third of the tibia, and being accompanied by an oblique fracture in the upper third of the fibula. In fact, the more oblique the fracture of the tibia, the more widely is it separated by the accompanying fracture of the fibula. It is in these extreme cases only that the modern practice of internal splintage is likely to replace the older method and yield better results. Lantern slides of such a case are shown as an example of how, by internal splintage, the shape of the bone may be almost perfectly restored. The operation is much more simple on the tibia than on the shaft of the femur, because of its superficial position and easy exposure.

The Different Behaviour of the Screws in the Proximal and Distal Fragments.

In one case two steel plates were placed over the fracture in the lower third of the tibia and maintained in position with four screws. The wound was then closed and the leg immobilised in a plaster splint. Union was by first intention. A skiagraph taken shortly after the operation, through the plaster splint, showed the two plates and the four screws which hold them more or less parallel to each other. The next slide, made from a skiagraph taken about six weeks after the operation, showed that one plate had become loose and had tilted, making one screw tear its way through the medulla of the bone, so that it is no longer directed similarly to the other screws. Three or four months later it became necessary to remove the plates, when it was found that both the screws distal to the fracture had become loose and were easily lifted out of their sockets. Whilst the two screws proximal to the fracture were tight and had to be unscrewed. This is a curious observation and suggests possibly that some constitutional disturbance, possibly vascular but more probably nervous and trophic, has weakened the metabolism, lessened the resistance and capability of repair in the fragment which has been severed from its vascular and nervous supply by the line of fracture. That such a condition should be brought about by the fracture is by no means surprising if one pauses to think about it. In fact it is almost as unreasonable to expect perfect vascular and nervous influences in an amputated limb as in a fragment separated by fracture from its parent bone. Hence, in applying internal splints to a broken bone it must be expected that they will hold less well in the fragment which has been separated from its vascular and nervous connection than in the other. Moreover, reparative changes in the "insulated" fragment may lead to some superficial necrosis round the screw. In the case narrated the limb remained in plaster after operation for five weeks, during which period there was absolutely no pain, discomfort, or fever. The wound healed, but later opened at one part where a sinus formed leading to the plates. About four months after the operation the plates were removed, and about a month after that whilst at a convalescent home a spicule of bone, about as long as the screw, which came from the lower and distal fragment. I would suggest that this was either a case of quiet and relatively aseptic necrosis in the lower fragment due to its isolation from its nervous and vascular supply, as the upper fragment underwent no necrosis; or the *Staphylococcus albus*, which has so much to do with the repair of a part, may have been concerned in the necrosis of a layer of bone in the insulated fragment.

October 12th, 1908.

BAILLIÈRE, TINDALL & COX announce the publication of Dr. T. Axenfeld's work on 'The Bacteriology of the Eye,' translated by Angus Macnab, B.A., B.Sc., M.B., Ch.B., F.R.C.S., Chief Clinical Assistant to the Royal London Ophthalmic Hospital. This valuable work contains 105 illustrations, mostly coloured, and the price is 21*s.* net. The size is large octavo, and the number of pages is 425.

WITH MR. SHERREN AT THE LONDON HOSPITAL.

Two Cases Illustrating Points in the Surgery of the Biliary Passages.

Movable Kidney and Gall-stones.

This patient illustrates one of the complications of movable kidney to which very little attention is paid—disease of the gall-bladder and biliary passages. She is æt. 42 years, and first came under my care twelve months ago. She had then been suffering for eighteen months from attacks of pain, starting in the right hypochondrium and passing round the right side to the shoulder; these attacks were accompanied by vomiting, but she had never been jaundiced. I had no doubt that they were caused by a stone in the gall-bladder. There was slight rigidity of the upper part of the right rectus muscle and deep tenderness midway between the umbilicus and the ninth costal cartilage. The right kidney was movable.

I opened the abdomen through the right rectus muscle, and, after tying off many adhesions around the gall-bladder and first part of the duodenum, removed a solitary stone from the gall-bladder and drained it by tube. I removed the tube at the end of a week, and bile flowed from the sinus for fourteen days, and then stopped suddenly, as not infrequently happens in these cases. She left the hospital soundly healed twenty-three days after operation.

I heard no more of her until March, when she came up having had attacks of pain in the right side, passing downwards to the groin, and accompanied by frequency of micturition, the urine during and for a day or two after the attacks, containing blood. The first attack came on six weeks after she left hospital; she had had five in all, each one more severe than the preceding. These attacks seemed to me to be due to her mobile kidney. X-ray examination revealed no stone. A fortnight ago I fixed her right kidney, and she is now healed and waiting until her three weeks' confinement to bed is over.

Many methods have been recommended to fix a mobile kidney. Three points should be remembered if success is to be obtained: (1) The kidney is to be fixed in position, so that the direction of its vessels and the ureter is normal; (2) the fibrous capsule must be stripped from its posterior surface; (3) the stitches which unite the kidney to the posterior abdominal wall until adhesions have formed, must pass through the reflected capsule and not through the kidney itself.

The influence of movable kidney on the biliary passages is liable to be overlooked. There is no doubt that obstruction to the outflow of bile may be caused by the traction of the mobile kidney on the duodenum. This may be complete, leading to an obstructive jaundice, and you have all heard me say that an obstructive jaundice unassociated with attacks of colic and accompanied by a distended gall-bladder in a young woman is usually due to a movable kidney. Fixation of the kidney will cause this to disappear. But sometimes there are, in addition, gall-stones, as in a woman, æt. 26 years, upon whom I operated three years ago, fixing her mobile kidney and removing several small stones from her common bile-duct. She has remained well ever since. In this case there was no doubt about the presence of stones, as there had been attacks of biliary colic.

The biliary symptoms associated with mobile kidney fall into two groups: (1) Those with obstructive jaundice, which may be due to the kidney alone or be associated with stones in the common duct; (2) those in which the biliary obstruction is too slight to cause jaundice, but has been the predisposing cause to cholecystitis and the formation of gall-stones, as in the present patient.

Stone in the Common Bile-duct.

This patient, who is now ready to leave hospital, is a typical example of the result of a stone in the common bile-duct.

She is a married woman, æt. 54 years, and has had eleven children. For the last six years she has had attacks of biliary colic; at first the intervals between the attacks were about a year, but during the past twelve months the attacks have occurred nearly every week. Jaundice was not present until about twelve months ago; it has been more or less marked ever since, becoming deeper after an attack and then gradually diminishing until the next attack. With the attacks she has had "shivering and fever," and has been losing weight. When I saw her a

month ago she was deeply jaundiced and passing clay-coloured stools and bile-stained urine. There was rigidity and deep tenderness in the gall-bladder region, but the gall-bladder was not distended. I had no doubt that for the past twelve months she had had a stone in the common bile-duct playing the part of a ball valve. At the operation, three weeks ago, after tying off many adhesions I found a thick and shrunken gall-bladder containing stones. As it was thick and shrunken I removed it. The common bile-duct was dilated and contained a rounded calculus in its supra-duodenal portion. This I removed through an opening at the junction of the cystic and hepatic ducts. I closed the opening almost completely and put a drain down in the duct. Bile flowed for a few days and has now quite ceased, and the wound is healed. The jaundice has practically disappeared, the fæces are of their natural colour, and the urine contains no bile.

Considerable difference of opinion exists with regard to the treatment of gall-stones. I, in common with the majority of surgeons, hold that operation is indicated if the gall-stones are giving rise to symptoms. Delay, as in this case, necessitates the performance of a much more severe operation, or may lead directly to the death of the patient from suppuration in the gall-bladder, biliary passages, and peritoneum, or indirectly from malignant disease. If there are symptoms pointing to the presence of gall-stones, the sooner the patient is put under the care of the surgeon the better.

Carcinoma of the Stomach.

Three or four months ago this patient, a woman, æt. 63 years, noticed that she had a distaste for food—a most important symptom in patients past middle life. Shortly after, she had pain of a gnawing character, coming on about an hour after food and sometimes waking her at night. She vomited for the first time a few days ago, bringing up a little blood. These symptoms point unmistakably to carcinoma of the stomach, and, as there is no evidence of dilatation, to carcinoma of the body of the stomach, probably of the lesser curvature or posterior surface. The test meal gave confirmatory evidence; there was an absence of free HCl. I propose to operate to-morrow.

The surgery of carcinoma of the stomach can be stated in a few words. If the growth is operable, that is, if it is free from surrounding structures, the affected glands can be removed, and there are no deposits in other organs, it should be widely excised together with its lymphatics, and the continuity of the alimentary canal restored. If the growth is inoperable and causing pyloric obstruction, gastro-jejunostomy will relieve the patient. If the irremovable obstruction is at the cardiac end, gastrostomy or jejunostomy is to be considered. If situated elsewhere, for example on the lesser curvature and irremovable, the abdomen should be closed without interference. Gastro-jejunostomy, which gives such striking results in chronic ulcer in this situation, is in these cases useless; it does not relieve the symptoms or prolong life.

The Treatment of Fractures of the Patella.

This woman, æt. 40 years, slipped four days ago in trying to save herself from falling and fractured her right patella by overaction of her quadriceps muscle. There is only one treatment that will certainly produce bony union in transverse fracture of the patella—open operation. By no other means can the fractured bony surfaces be brought into apposition. If you look at this X-ray plate you will see that the lower fragment is tilted so that its fractured surface looks forwards instead of upwards, and its cartilaginous surface towards the fractured surface of the upper fragment. In addition, you will see when I operate to-morrow that the aponeurosis which runs over the patella from the quadriceps muscle is tucked into the joint, hence bony apposition is impossible.

There are several important points. First, What are the indications for operation? All patients with transverse fracture of the patella with separation, in whom there are no general indications contra-indicating operation, should be told that the only method by which a sound and freely movable limb can be obtained is by open operations; they should have everything laid before them and left to choose the course they think fit. In men in whom a perfectly movable and reliable limb is necessary, operation should be urged. The next point is, When should operation be done? The sooner the better. I have wired recent fractures of the patella as early as four hours after infliction. No time should be lost. A flap should

be turned up, giving a free exposure of the fragments and the torn capsule. All clot should be wiped out with sterile gauze or washed out with sterile salt solution and the bone drilled and united by two silver wires, the torn capsule sutured with catgut and the wound closed without drainage. After being dressed it is well bandaged over plenty of cotton-wool. I never use a splint. The patient may get up in a chair, keeping the leg extended, as soon as convenient. As soon as the wound is healed and the stitches are removed, passive movement and massage is started and the patient allowed to walk. As a rule they are able to walk out of hospital from the fourteenth to the seventeenth day and should be able to resume work within six weeks of operation.

The wire remains in and gives rise to no trouble. I have only had to remove it once, from a man who fell and struck his knee three months after I had wired his patella. He then noticed the wires and wished them removed. This I did for him and he was content.

Brachial Birth Paralysis.

This boy, æt. 6 years, is the first-born child; labour was long and forceps used. When a few weeks old his mother noticed that he did not move his right arm so well as his left. No systematic treatment has been carried out, and he has been sent to me with a view to operation.

You will notice at once that the right upper limb is in the position typical of Erb-Duchenne paralysis. The arm is held close to the side and the forearm is extended and pronated. As you see, he makes no attempt to abduct the arm or to flex or supinate the forearm. As is always found in these cases, the pectoral muscles are contracted, rendering full passive abduction of the arm impossible. There is, as usual, no sensory change.

From the present examination I cannot say what, if any, nervous recovery has taken place. But, from the history, that no tenderness was present during the first few weeks of life, I suspect that recovery of the nerve affected has taken place. This we can soon settle by testing his electrical reactions.

You see that the affected muscles all react readily to stimulation with the interrupted current, so that the nerve lesion has been recovered from. Yet the limb remains in an abnormal position, and the boy does not use it. This is by no means an uncommon condition. This failure of restoration of function, in spite of nerve recovery, is due to lack of efficient treatment in the early stages. No attempt has been made to prevent the contracture of the opposing muscles and to keep up the nutrition of the affected ones, and encourage movements as the muscles again receive their innervation.

The treatment of this particular case is passive movement to overcome the contraction of the opponent muscles, and daily massage and stimulation with the interrupted current with systematic attempts at voluntary movements. In the intervals of treatment the limb is to be put up with the paralysed muscles relaxed. If this is conscientiously carried out there is no reason why recovery should not take place.

The treatment of birth paralysis is simple when the patients are seen early. The limb is put up with the paralysed muscles relaxed. A pad of cotton-wool is put in the axilla and the arm and forearm then bandaged to the body over cotton-wool, the forearm being kept supinated. The limb should be taken down daily for massage and should be treated on these lines until the restoration of voluntary power ensues or it becomes evident that the nerve division is complete. If at the end of three months no recovery has taken place, the electrical reactions of the affected muscles should be tested under an anæsthetic; if they are those of degeneration, operation should be performed. Recovery apart from operation may be expected, if the patient is treated on these lines, in over 70 per cent. of the cases.

Facial Paralysis Following Mastoid Operations.

This young man, æt. 26 years, has had the radical mastoid operation performed on both sides. Before this he had had three operations on the right side, and after the last of these, twelve months ago, the facial paralysis was noticed. Seven months ago he was sent to me as a suitable case for facio-hypoglossal anastomosis, and I was told that the "reaction of degeneration" was present. I took him into hospital and found complete flaccid facial paralysis on the right side, but the reactions were not what I considered those of degeneration, but were those I described as typical of a certain degree of incomplete division.

I gave a good prognosis and said that in all probability recovery would ensue without operation. He has been treated by massage and stimulation, at first with the constant current, later with the interrupted, and I have been seeing him every month. As you can all see now, it is extremely difficult to say, on superficial observation, which side of the face has been affected. But if you will look at his forehead you will notice that the frontalis muscle is paralysed; all the other muscles supplied by the facial nerve are acting well. Those muscles which are acting now react to stimulation with the interrupted current. This is the condition met with in most cases of injury to the facial nerve due to the mastoid operation. The nerve division is rarely complete, hence nerve anastomosis is rarely necessary.

Varicose Veins.

I have recently been writing to all those patients upon whom I have operated for varicose veins. This man has come up in response to my letter.

The operation that I perform in the usual case with varicose internal saphenous vein and its branches, is the removal of about an inch of the internal saphenous vein in the upper part of the thigh in the region of the saphenous opening. This is necessary in every case in which operation is indicated because symptoms are present, and in many, is all that is required to relieve the patient of all the troubles due to the enlarged vein with incompetent valves. But where the vein is markedly enlarged, and there are pouches adherent to thinned skin such as are not uncommonly found in the lower third of the thigh and upper third of the leg, these should be excised. When the external saphenous is affected it should be treated in a manner similar to the internal.

I have found this operation successful beyond my expectations. The patient who has come up this afternoon was able to go back to work within four weeks of operation, and has had no pain or inconvenience since. Seven years before I operated he had had a mass of varicose veins removed from the inner side of the leg without any appreciable relief to the pain. When I saw him two and a half years ago the internal saphenous was enlarged from the saphenous opening downwards, and an impulse on coughing could be felt right down to the foot. I did the operation I have described with the result you all can see.

Indications for Operation on Varicose Veins.

We will for a minute put on one side those patients who have to have their varicose veins removed because they wish to enter one of the public services; the presence of an enlarged vein in these cases is the indication. But in others I hold that this is not a sufficient indication. Operation is not indicated until symptoms arise, and in many cases these are so slight that they can be kept in abeyance by wearing a light *crêpe* bandage. The most important indication comes first, that of thrombosis. I always operate on patients with thrombosis of the internal saphenous vein, and consider it extremely unwise, to use a mild term, to leave these patients without operation. In the first place, the danger to life is not trifling; not long ago a patient in this hospital who was admitted with thrombosed internal saphenous vein and was awaiting operation suddenly died of pulmonary embolism, and I can remember several such cases. Then the period of illness is much diminished; under the old method of treatment at least six weeks in bed were necessary, and it not uncommonly happened that at the end of this time as soon as the patient put the affected leg to the ground fresh thrombosis occurred.

These cases should be operated on as soon as possible. In operating, the skin in the region of the saphenous opening may be cleaned up before operation but not the skin in the region of the thrombosis. After the vein has been exposed at the saphenous opening and about an inch removed, this wound is closed and the skin over the affected part of the vein cleaned up; the whole of the affected portion of the vein is excised, and it is often necessary to remove the skin covering it as well. The result of this operation is excellent; in three weeks the patient is able to get up, free not only of the thrombosis but of the varicose veins.

The next indication of importance is, I consider, ulceration. A chronic ulcer of the leg associated with the presence of varicose veins will heal if the limb is put at rest and elevated, but if the veins are not treated recurs on resuming the erect attitude. When a chronic ulcer of leg is present with varicose internal saphenous veins I consider that

the operation which I have described should be carried out; the results have been very good. In one patient, a girl, æt. 26 years, upon whom I operated four years ago, the ulcer had been present off and on for eighteen months. I did the usual operation and saw her quite well a week ago; the ulcer had healed before she left hospital, and has not broken out since.

When pain and swelling of the leg is present operation should be performed, if this is not controlled by a *crêpe* bandage applied before the patient puts the foot to the ground in the morning and worn all day.

Multiple capillary varicosities; most often seen in women, are not suitable to operation in this way. I consider that the pain and all the symptoms are due in the cases we have been discussing to incompetence of the valve, and it is for this reason that the operation is successful.

Carcinoma of the Cæcum.

This man, æt. 54 years, was admitted to the hospital five weeks ago as a case of appendicitis with abscess. He had had two attacks of pain in the right iliac region which were not accompanied by vomiting. The first attack was six months ago, the last three days before admission. On admission he had a temperature of 100° F. There was a rounded, fixed and nodular swelling in the right iliac fossa, with slight deep tenderness over it, which, from its physical signs, might have passed for a chronic appendix abscess surrounded by much fibrous tissue. But there were several suspicious features in the case. He had had looseness of the bowels for several months, and had on several occasions passed a little blood in the stools. On examining the abdomen generally, dilated coils of small intestine were seen. These points, with the feel of the tumour, made me diagnose carcinoma. Three weeks ago I explored the tumour and found a fixed mass which was undoubtedly malignant. Through the wall of the cæcum I was able to feel an ulcer with hard edges, and on inspection growth was seen spreading under the peritoneal coat. Radical operation was out of the question, so I divided the ileum close to the cæcum and closed both its ends, and performed lateral anastomosis between the ileum and ascending colon. This has relieved his symptoms, and he will soon be able to leave hospital.

Carcinoma of the cæcum presents difficulties in diagnosis. It is rarely seen early enough for complete removal. This patient is a type of the disease as we most often see it, diagnosed as are so many others, as appendicitis.

Great care is necessary in the differential diagnosis, we have to bear in mind, in addition to appendicitis, actinomycosis and ileo-cæcal tuberculosis. A diagnosis of malignant growth must never be made without conclusive evidence. I have known on two occasions an appendix abscess diagnosed as carcinoma of the cæcum, much to the distress of the patient, and it required considerable trouble to convince one patient that the first diagnosis was not correct and that no cancer was present. In one of these cases which was under my care there was no obstruction of small intestine and no passage of blood; the swelling would have fallen in with the diagnosis of carcinoma of the cæcum, but the history and the other signs were against it.

In the treatment of carcinoma of the cæcum, removal of the cæcum and its lymphatics should be carried out, if possible. If this cannot be done, the ileum should be divided as in the present case, and anastomosed to the colon as close as is safe to the growth.

October 12th, 1908.

We have received from Burroughs Wellcome & Co. a sample of their "Hemisine" (1 in 1000). This is suitable for the preparation of aseptic solutions. When employed undiluted, "Hemisine" 1 in 1000 solution may be sterilised by boiling immediately before use. When dilution of fluid preparations of "Hemisine" is required, the diluting fluid should be normal saline solution, except in the case of eye-lotions, when sterile distilled water is preferred. "Soloid" Sodium Chloride, 0·17 grm., which is issued together with "Hemisine" 1 in 1000, affords a ready and convenient means of preparing normal saline, one "Soloid" product being dissolved in 25 c.c. (7 fl. drm.) of sterile (boiled) water. To obtain a dilute sterile solution with "Hemisine" 1 in 1000 solution or with "Soloid" "Hemisine," the diluting fluid should be raised to the boiling-point, the "Hemisine" then added, and the boiling continued for two or three minutes. An interesting and readable account of the use of "Hemisine" is issued by Burroughs Wellcome & Co. for medical men.

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THE TECHNIQUE OF CANCER OPERATIONS, WITH REFERENCE TO THE DANGER OF CANCER INFECTION.

By CHARLES RYALL, F.R.C.S.,

Surgeon to the Cancer Hospital, Brompton, S.W.

FROM a surgical point of view cancer is a spreading infective process, and the cancer-cell contains the elements of infection. This fact can be better appreciated by considering the cancer process as a whole. The primary cancerous growth is the primary focus of infection. From this point the disease tends to spread and disseminate. The cells multiply indefinitely. They invade and infect the tissues around the primary growth; they infect the nearest lymphatic glands, and they may be carried to distant parts of the body. They are capable of independent existence apart from the primary growth, and wherever they come to rest they are capable of reproducing themselves and starting fresh cancerous growths. These secondary cancerous growths may in time become foci of further infection.

Resemblance to bacterial infection.—These peculiarities of the cancer-cell very much remind us of certain infective bacteria. The latter may multiply indefinitely, and may infect the tissues around the primary focus of infection; they may also infect the nearest lymphatic glands, or they may be carried to other parts of the body, and thereby set up further foci of infection. The primary cancerous growth may be chronic in its nature, without any tendency to spread, and the same with the primary focus arising from infective bacteria, which may remain localised. On the other hand, some small cancerous growths have a tendency to early formation of metastases, just in the same way that multiple pyæmic abscesses may readily follow an apparently insignificant focus of septic infection. Also the rapid brawny infil-

tration of some primary growths may be compared to the diffuse spreading cellulitis resulting from bacterial infection. These points help to demonstrate that cancer is a spreading infective process not unlike other infective processes; that the cancer-cell within the body is possessed of independent existence; that it contains the elements of infection, and that it can give rise to fresh cancerous outbreak in any part of the body, whither it is carried by the lymph or blood-stream, just in the same way as wandering bacteria are capable of forming secondary infective foci. In the treatment of cancer we have, therefore, to deal with an infective process, and we must seriously consider the question of cancer infection. Cancer infection is the implantation of cancer-cells from cancerous growths into tissues unaffected with the disease, and the occurrence therefrom of a fresh cancerous outbreak.

Types of cancer infection. — Contact infection is the outbreak of fresh cancerous growth in healthy tissues which have been in contact with infected tissues. It is most frequently seen in the peritoneum, but is also found in other parts, especially the pleura, vulva, lip, mouth, and bladder. It is not so common as one would expect, and therefore one concludes that certain conditions are necessary or favourable for its development. When a cancerous growth invades the peritoneum it shows little tendency to break down or disintegrate like external growths. Therefore, any cells which are cast off are probably active, and readily capable of implantation, either on the opposing peritoneum, or in any stomata in which they get lodged. On the other hand, cancer of the lip, mouth, and vulva only occasionally gives rise to contact infection. Growths of these parts readily break down and ulcerate. The cancer-cells thus cast off are likely to be dead and inactive, and incapable of implantation. For cancer infection to take place it is probable that liberation of active cells and breach of surface in the opposing structures, to admit of implantation, are necessary. This would account for the occurrence of cancer infection in these parts, which are, as we know, subject to trauma, whereas in cancer of uterine cervix, a part protected from injury owing to its position, contact infection of the vagina is almost unknown. Contact infection is also occasionally met with in the bladder, and, more rarely, in the alimentary tract. The fact that it is not of more frequent occurrence may also be explained by the readiness of growths in these parts to undergo necrosis and ulceration. *Distant implantation* is another common form of cancer infection. It is the development of cancerous growths in parts distant from the primary growth, as the result of cancer-cells being conveyed and implanted there. This is also most commonly met with in the peritoneum, where cells liberated from a cancerous growth get carried by peristalsis or the force of gravity, and become implanted in some distant part of the peritoneum. This form of infection is frequently found in the pelvic peritoneum, as the result of cancer-cell droppings from growths in the upper abdomen Not only the pelvic peritoneum, but the large peritoneal pouches on the outer sides of the ascending and descending colon are frequently the site of similar implantations in bed-ridden patients suffering from peritoneal cancer. The pleura is also the site of similar implantations. Another common form of distant implantations is found in the bladder, as the result of the presence in the upper urinary tract of villous papilloma, which is virtually a malignant growth. Distant implantations in the alimentary tracts are rare, and this is probably due to the same reason as the rarity of contact infection in these parts.

Operation infection.—In a recent paper ("Cancer Infection and Cancer Recurrence," 'Lancet,' November 9th, 1907), I drew attention to this danger, and enumerated a series of cases in support of my arguments. These cases demonstrated certain phenomena which occurred after operation, resulting not only in recrudescence of the disease, but, in some cases, causing aggravation of the original trouble. Operation infection may manifest itself in many ways. It may occur early, or it may appear late. It may manifest itself as a solitary nodule, from implantation of a small fragment of cancer, or it may be seen causing widespread disease as the result of diffuse cancer infection. It may appear in the neighbourhood of the growth or it may develop in healthy tissues cut into at the time of operation but far removed from the part primarily affected. *Suture-scar infection* is a very common occurrence. It is the result of cancer-cells being implanted by an infected needle or suture. I have seen it appear

as a solitary nodule in one scar, and I have also seen it in many. Sometimes it has occurred as an unbroken chain in the suture-scars, and on other occasions some scars have become infected and others have escaped. It may appear in the superficial part of the suture-track or in the deeper—wherever the cancer-cells happen to become implanted. Sometimes the entire suture-track may be the site of infection. In one case of mine the site of infection seemed to indicate how implantation had taken place. Nodules developed in four suture-scars, two on each side of the wound, immediately opposite one another. The nodules on one side appeared on the surface, and those on the other immediately beneath the skin. This seemed to indicate that the infected needle or suture, on being inserted, cleaned itself of cancer-cells, and thus caused surface infection; and then being re-handled by the surgeon, became re-infected, and when thrust through the skin-flap on the opposite side, it again cleaned itself and set up infection immediately beneath the skin. Infection of the entire suture-track is most frequently found in exploration or interference with abdominal cancer. I have only seen it appear when the suture is passed through all layers of the abdominal wall, including the peritoneum. This may be caused by sutures being infected with cancer-cells at the time of operation, but it is more probably due to cancer-cells free within the peritoneum escaping along the suture-track and thereby setting up infection. I have come across this form of infection in many parts of the body, but especially in connection with carcinoma of the breast, abdomen, rectum and glands of the neck. Cancer infection of suture-scars reminds us in some ways of the phenomena which occur as the result of infection following septic sutures. *Nodular infection* of the wound is also a fairly common manifestation. The appearance of nodules of infection within the field of operation was generally ascribed to the surgeon having failed to cut sufficiently wide of the disease, but there is no doubt that in many cases this is due to re-infection of the wound with cancer-cells which have been allowed to escape from the growth which is being removed or interfered with. It is no uncommon occurrence to see single or multiple nodules spring up after operation for the removal of a solitary growth of limited extent. In a case where I attempted to remove a solitary nodule from the chest wall I have seen fourteen nodules spring up after operation. The patient had her breast excised ten years previously, and though there had been several subsequent operations she had been free from disease for five years; hers was, therefore, a chronic condition. The interesting point in this case is that, in order to remove the nodule, a skin-flap was turned back, and the nodules which developed themselves after this operation only appeared on or beneath the skin-flap, and in the line of incision. None appeared outside the field of operation, nor were any perceptible beyond this for at least six months afterwards, up to the occasion of my last seeing the patient. This case illustrates, more or less, widespread cancer infection of the wound, but sometimes only a solitary nodule may appear within the field of operation, showing more limited cancer infection. As an example of this I may mention a case of abdominal hysterectomy for uterine cancer, where limited infection of the abdominal wound apparently took place, as was demonstrated by the appearance of a solitary cancerous nodule in the abdominal cicatrix some months after operation.

Diffuse or brawny infection of the wound.—This is the most distressing form of operation infection. It is not only met with following an attempt at radical operation, but it is also seen appearing after surgical interference with inoperable growths. I have no doubt that it is due to widespread dissemination of cancer-cells at the time of operation, with consequent widespread infection. It almost invariably appears early, and it is characterised by brawny induration and infiltration of the tissues within and around the field of operation. It is not only alarming in its extent, but also in the rapidity with which the disease seems to progress. This form of infection, in my experience, has been most frequently met with in connection with interference with malignant glands in the neck. It has occurred after curettage of a breaking-down cancerous gland, and it has also occurred after extirpation of glands moderately, as well as extensively, enlarged. In the cervical region I have seen signs of diffuse infection occur within a fortnight after operation, and within the course of another two weeks the whole side of the neck has been converted into one brawny cancerous mass. In many of these cases cancer infection was suspected at the time of

operation. The neck is not the only part where this form of infection has come under my observation. Following amputation of a carcinomatous breast I have seen similar diffuse infection occur. At first a few suspicious nodules appeared, but within eight weeks the field of operation and the surrounding part of the chest became one diffuse mass of brawny cancer. This was a case of a small carcinomatous tumour of short duration, but cancer cells were, unfortunately, known to have been set free at the time of operation, and thus infected the wound. I have also seen a similar form of diffuse infection occur in the structures surrounding the rectum following partial proctectomy. In this case cancer infection was also suspected at the time of operation. *Distant implantation* is that form of operation infection where cancer-cells are carried to and implanted in a part cut into at the time of operation, but more or less distant from the original growth. As examples of this I may mention infection of laparotomy wounds. I have already quoted a case where infection from cancer of the uterus showed itself in the abdominal cicatrix, but I have also seen the abdominal wounds in several patients infected from cancer of the large intestine. In one case, which is of interest, where the abdomen was opened through the right iliac fossa in order to remove the appendix, a small mass of malignant disease was found on the left side in connection with the pelvic colon. An attempt to remove this had to be abandoned, but nevertheless, manipulation of the growth had set free cancer-cells, and later this patient developed nodules of growth in the abdominal cicatrix. Another example of distant implantation occurred in connection with amputation of a cancerous breast. The wound was drained by an opening made through the posterior axillary fold, and later, in the drainage-tube cicatrix, a nodule of infection appeared. The same scalpel which had been employed for amputating the breast was also used for making the drainage-tube opening.

Operation technique.—Cancer-cells which escape from a growth during operation, and which find a resting place in any part of an open wound, may develop, and frequently do so, into fresh cancerous growth. It is the presence of this risk which demands special technique in cancer surgery. Therefore, unless we exercise the utmost care in performing cancer operations our efforts are rendered null and void. No matter how perfect our asepsis, and no matter how extensive the operation, cancer infection is likely to show itself unless proper precautions are taken. In all operations elaborate means are adopted to prevent septic infection of the wound, but in cancer operations, not only are these careful measures necessary, but others equally elaborate must likewise be adopted, in order to prevent cancer infection. Septic infection manifests itself early, at a period when the patient is kept under strict observation, and moreover, it sets up symptoms more or less acute which bring about its early recognition. Cancer infection, on the other hand, generally shows itself late, after the wound is healed, and all is thought to be well. The onset of its manifestation is insidious, and it is only by the mere bulk of cancer-cell growth that it can be recognised. Any failure to prevent cancer infection is a grave matter, as it is seldom that one has a second satisfactory opportunity of operating on a cancerous infected area with a hope of a favourable result. In order to study the steps necessary for the prevention of cancer infection let us consider how it may occur. As a first step cancer-cells must be liberated from cancerous growth, in which they are more or less pent up. This may occur through: (1) Exploratory incisions into cancerous tumours; (2) cutting into infected tissues during operation; (3) rupture of infected lymphatic glands, or infected lymphatic vessels; (4) tearing or lacerating cancerous growths; (5) piecemeal removal of cancerous growths; (6) roughness in manipulation, and (7) curettage. The cells liberated by any of these means may implant themselves in the wound or some raw surface, and thus infect tissues as yet unaffected with the disease. They may contaminate instruments or other appliances used in the operation, or infect the surgeon's or assistant's hands, and thereby give rise to cancer infection. The operation may be completed, the wound may heal, and the result appear everything that could be desired. Nevertheless, the elements of infection are left behind, only to manifest themselves later. Therefore no operation, however elaborate, however extensive, nor however skilfully carried out can hope for a successful issue unless the entire disease be removed, and cancer-cells prevented from escaping from the growth and implanting themselves in the wound.

Exploratory incisions into tumours.—Exploratory incisions into tumours have frequently to be made in order to settle the question of diagnosis. If malignant disease be present a more or less extensive operation is indicated, and if this operation be carried out at once grave danger is incurred unless precautions are taken to avoid cancer infection. When a growth is cut into cancer-cells escape from the tumour, where they had heretofore been held fast. They float out in the escaping blood, and infect or cling to the surgeon's hands, scalpel, other instruments and towels used for the operation, and anything else with which they come in contact. This is not mere imagination, as it was proved to my satisfaction with the aid of a microscope. If one, therefore, proceeds to excise a cancerous tumour without making further preparations, these free cancer-cells are likely to infect the freshly incised tissues. It is, therefore, of the utmost importance to guard against the danger of implanting these cells into any fresh wound. Failure to take this precaution was, I am afraid, the cause of rapid and extensive infection in one of my earliest operations for breast cancer. In dealing with a cancerous growth into which an exploratory incision has been made it is, of course, safer to suture the incision and wait until healing has taken place before carrying out its removal. This gives an unbroken surface for the subsequent operation, and cancer-cells being pent up, it obviates the risk of cancer infection, and, moreover, prevents cancer-cells being squeezed out during manipulation of the tumour. Unfortunately we cannot often adopt this measure, because delay in dealing with cancer is a serious matter, and moreover, postponement necessitates a second anæsthetic, and in the patient's opinion, this is a second operation. Where excision is immediately carried out I adopt the following precautions :

(1) The exploratory incision is securely closed, after seeing that all hæmorrhage has been checked.

(2) After closing the wound steps are taken to prevent any escape of blood between the sutures during subsequent manipulation.

(3) All instruments, sponges, ligatures, and sutures which have up to this stage been used are promptly discarded, or else re-sterilised before being used again.

(4) Fresh cloths or towels are employed for isolating the area of operation and those previously used are laid aside.

(5) All lotion and lotion bowls are considered as being infected and a fresh lot are supplied.

(6) After closing the wound the skin in the neighbourhood of the exploratory incision is washed free of all blood, and then thoroughly prepared by further washings with antiseptics, just as carefully as it is prepared preliminary to any operation.

(7) The surgeon's hands, and those of his assistant, are re-washed and scrubbed, and prepared with the minutest detail, in a similar manner to that carried out at the commencement of the operation.

I take just as careful precautions as if some virulent bacteria were let loose by the exploratory incision. The arrest of hæmorrhage is important, because as long as bleeding persists cancer-cells are likely to ooze out with the blood. If oozing persists, even after closure of the wound, manipulation of the tumour may squeeze out cancer-cells. Closure of the wound is equally important in order to prevent cancer-cells escaping during manipulation, and it is this manipulation during the subsequent steps of the operation which renders mere suturing, even if followed by collodion dressing, insufficient to prevent the escape of cancer-cells. I have therefore resorted to a measure which renders manipulation of the tumour perfectly safe, and which has been found most satisfactory in practice. After carefully checking all bleeding, deep "through-and-through" sutures are inserted for the purpose of closing the wound, but before they are secured a long pad or roll of gauze is laid along the line of incision, and the sutures are tied over this. The sutures bring the incised edges firmly together; and moreover, if any oozing persists, and blood escapes or gets squeezed out during manipulation it soaks into the gauze pad. Free handling of the growth can thus be carried out with safety. I regard this as an important step, as I fear that insufficient care in carrying it out was responsible for the severe infection which occurred in another of my cases. When the exploratory incision had been sutured, and during the subsequent operation, I discovered that blood was being squeezed out by my manipulations. That the instruments, etc., used in these exploratory incisions, and also the surgeon's hands, may become carriers of cancer-cells is not

mere imagination. This has been proved to my satisfaction with the aid of the microscope. Numerous experiments have been made, and though a positive result was not obtained in all, yet the evidence was convincing as to cancer infection being conveyed by this means. A scalpel which had been used for mere incision into a cancerous growth has, as a result, cancer-cells almost invariably clinging to the blade, and I think I can safely say that cancer-cells are always liberated into the blood escaping from the incision. The necessity of careful attention to the hands was well demonstrated to me in a case of exploration of mammary tumour. They had been well rinsed until apparently free from all traces of blood. Nevertheless, the microscope proved that this was insufficient cleansing, as cancer-cells were found lodging under my finger-nails.

Removal en masse.—As long as cancer-cells are pent up we are operating under the best conditions, and therefore when operating for cancer the infected tissues should always be removed *en masse.*

When this cannot be done an attempt should be made to carry it out as closely as possible. If we could always ensure removal of a shell of healthy tissue entirely surrounding the diseased area, and therefore all cancer-cells enclosed within this, operation infection would not occur. One should, therefore, draw an imaginary line round all structures which it is proposed to remove, and excise them *en masse.* The removal of the nearest lymphatic glands is considered a necessary step in cancer operations, because they are presumed to be infected, and if the latter be correct one must, therefore, consider the lymphatic channels leading from the growth to these glands as also a site of infection. The glands and these channels must consequently be removed *en masse* with the cancerous growth and its surrounding structures, and where it can possibly be avoided these lymphatic channels should never be cut or torn across. In removing glands it is equally important that they should be excised *en masse,* along with the surrounding fat and connective tissue. It is unscientific to accomplish their removal by dissecting them out one by one from the bed of tissue in which they lie. If they are infected, then their intercommunicating lymphatic vessels must also be considered as being infected. Therefore, unless the glands are removed *en masse,* as described, these vessels get torn across, and may permit of the escape of cancer-cells. I have seen cancer infection of the wound follow the removal of a few slightly enlarged glands in the neck, and the only explanation I could offer for the occurrence of this was, that in dissecting the glands out one by one, cancer-cells had been liberated by my tearing across some infected lymphatic channel. Moreover, removal *en masse* renders more complete excision possible, as by removing the surrounding fat and connective tissue small glands which otherwise might be overlooked are also removed. Again, this complete excision of the glands and the peri-glandular structures minimises the risk of lacerating or bursting glands during removal. In the neck the old plan of dissecting out a chain of glands was a difficult and unsatisfactory measure, and generally fraught with considerable danger of cancer infection owing to the tendency of cervical glands to undergo early softening, whereas now, in many cases, operation can be accomplished with comparative ease and less danger by excising the fat, fascia, and glands in one mass.

Roughness of manipulation.—Roughness of manipulation during operation is, I believe, accountable for many cases of cancer infection. Unless one exercises the greatest gentleness cancer-cells may be squeezed or forced out of infected channels or tissues; the site of disease may become torn or otherwise damaged, and give rise to cancer-cell dissemination. In one of my cases already mentioned, where the laparotomy wound became infected during the performance of abdominal hysterectomy, I am convinced that this occurred as the result of the malignant uterus being torn during operation and thus allowing of the escape of cancer-cells. From my own experience, extreme gentleness of manipulation is called for more frequently in operations for the extirpation of cancerous glands of the neck than for any others. I have mentioned that these glands show an early tendency to soften and liquefy, and therefore, removal without rupture of one or more of them is, in many cases, only accomplished with the greatest care in dissection and gentleness in manipulation. In my experience such severe cases of cancer infection have followed accidental laceration or rupture of glands removed from these parts that I regard this region as being particularly dangerous. Even when exercising the greatest care accidents will sometimes happen, when the surgeon will cut into or tear across a part affected with cancer. It is a most unfortunate accident with which to be confronted, as implantation of the escaping cancer-cells may render the operation futile. Should such an accident occur one must endeavour to frustrate the dissemination of cancer-cells as best one can. Both the infecting area and the area of infection should be attended to immediately. In these cases I recommend the application of the actual cautery to the malignant

growth which has been incised or torn, then drying, and afterwards swabbing the possibly infected area with 1 per cent. formalin solution, and a pad soaked in the same solution finally placed and kept in contact with the infecting area. The surgeon should then discard the instruments in use and have a fresh supply, and at the same time prepare his hands anew. Where the infecting area can be sutured over without interfering with the further steps of the operation, I advise that it should always be carried out. The modern operation of vaginal hysterectomy furnishes a very good example of roughness of manipulation, which should be avoided. Cancer-cells are liberated from the cervix, which is seized and lacerated with vulsella forceps, and during subsequent manipulation these cells become disseminated and implanted in the wound left after operation. The majority of these cases develop recurrence within the field of operation, and though a certain proportion escape, nevertheless, it is more by good fortune than by good surgery that a satisfactory result is ever obtained. The curette and sharp spoon are very useful instruments for dealing with cancer. Where the disease is degenerating and breaking down, and the removal of necrotic material is advisable, the use of the curette or sharp spoon is invaluable. It should never be applied so vigorously as to get beyond the area of the growth, and thereby open up uninfected tissues, as these latter only become implanted with cancer-cells carried there by the instrument. In one of my own cases, mistaking a breaking-down gland in the neck to be of innocent origin, I used the sharp spoon somewhat too vigorously and carried it into healthy tissues beyond the gland capsule. Unfortunately the gland was the site of carcinoma, with the result that implantations were made into the healthy tissues which I had opened up. This patient developed rapid brawny cancer infection of the neck, and was speedily reduced to a deplorable condition as the result of an unfortunate mistake. My object is to draw attention to the importance of cancer infection during operation. Although it is fully appreciated by many of those who see much of cancer, nevertheless I do not think that this is so with the profession on the whole. Many a case of cancer recurrence is due, not so much to the surgeon failing to cut wide of the disease, as it is to his faulty technique in allowing cancer infection to take place during operation. I am much indebted to all those who demonstrated for me the presence of cancer-cells in washings taken from instruments, hands and operation wounds, and I fully appreciate the amount of labour and skill which it entailed, but I am particularly indebted to Dr. A. Paine and Dr. D. Morgan, of the Pathological Department of the Cancer Hospital, who were the first to carry out this work for me.

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PROLAPSE OF THE UTERUS.

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In choosing prolapse of the uterus as a subject I felt that it was one which was of general interest, and which has always caused me much thought on account of the constant difficulties arising in the treatment of it. I always look upon severe degrees of uterine prolapse as conditions which tax our ingenuity to the utmost, whether we treat them from a palliative or an operative point of view. They also appeal to us all from the point of view of the patient, for apart from the constant pain of cancer, the urinary dribbling of a vesical fistula, or an inguinal anus, I cannot conceive a more miserable condition to be in than that which accompanies a complete procidentia of the uterus.

The name "prolapse of the uterus" implies a descent of that organ from its normal position in the pelvis, whereby it travels down the vagina, everting the vaginal walls as it descends. But this by no means describes the condition of affairs as we see them clinically, for the uterus and vagina are not the only organs displaced. The bladder comes down too with the connective-tissue and fat around it, and naturally the peritoneal coverings of these organs must also be displaced. The question naturally arises, then, whether we are correct in speaking of prolapse of the uterus when it is only a part of a much more general displacement. The researches of Hart and Barbour and others have made this question quite clear, and the answer is that we have to deal with a descent of all the movable structures in the pelvic floor. Strictly speaking the uterus is only a portion of the pelvic floor, because it forms the roof of the vagina, and therefore the uterus only descends because it is a part of the pelvic floor. Hart and Barbour describe the pelvic floor as consisting of a movable or pubic segment and a fixed or sacral segment. The pubic segment comprises the retro-pubic fat, the bladder and urethra, the anterior vaginal wall and the uterus, whilst the sacral segment includes the posterior vaginal wall, the

rectum, the perinæal body and the levator ani muscle with its fascial coverings. The mobility of the pubic segment is easily demonstrated in labour when the bladder, urethra and anterior vaginal wall are drawn up above the pubes over the advancing fœtal head, whilst the fixed sacral segment is either crowded into the sacral hollow or simply stretched, not really displaced. In descent of the uterus and anterior segment we find that the posterior vaginal wall also becomes movable, for it descends with the uterus and becomes everted like the anterior wall. This description of the pelvic floor is easily demonstrated and cannot but be believed as correct, and if so we must regard the name "prolapse of the uterus" as a useful clinical title, but incorrect as far as it is a definition. Strictly speaking we should call the condition "hernia of the pelvic floor," for it is a *true hernia*. It is a true hernia because it has a definite road to travel, namely, the space between the sacrum behind and pubes in front, and the levator ani on either side. It is a hernia because it has definite coverings, namely, the bladder, urethra, vaginal walls, uterus, etc. It is a hernia because it has a peritoneal sac inside, and because it has contents, namely, small intestine. Further, we shall see that the uterus, though a part of this hernia, has nothing to do with its causation, causes none of its symptoms and is not even the most important organ concerned. If symptoms are any guide the bladder is much more important than the uterus in this sacro-pubic hernia.

In order to understand the causation of uterine prolapse we must first have a clear understanding of the normal supports of the uterus and of the movable segment of the pelvic floor, especially the bladder. Our views upon this point have undergone considerable change recently, owing to the anatomical researches of Thompson, Keith, Paterson, and Derry, which have been very ably put together and reviewed by Fothergill in a recent paper read before the Obstetrical Section of the Royal Academy of Medicine. We must, however, consider the older views before the new. We have been in the habit of saying that the movable structures of the pelvic floor are held in position by: (1) the uterine ligaments; (2) the integrity of the sacral segment, especially the levator ani and its fascia; (3) the intra-abdominal pressure; and (4) the size, weight, and consistence of the uterus. To those of us who are in the habit of performing uterine operations it is clear that the ligaments of the uterus have nothing to do with that organ's maintained position, for none of them are tense structures, and none of them prevent movement of the uterus in all directions. The uterus can be pulled down to the vulva quite as easily with intact ligaments as it can when they are divided. Secondly, it is quite common to see a complete laceration of the perinæum into the rectum, with consequent division of all fibres of the levator ani in the perinæum without *any descent of the uterus or bladder*. Thirdly, an increase of abdominal pressure caused by straining at stool, chronic cough, or hard work will not cause a prolapse in a healthy individual who has not been pregnant. Fourthly, no increase in size or weight or alteration in the consistence of the uterus has anything to do with prolapse. In fact the larger the uterus, as in the case of fibroid tumours, the more difficult is it for the organ to descend through the space which is open to it. Further, the operation of vaginal hysterectomy administers the *coup de grâce* to these theories, because we may divide the vaginal walls, perinæum and uterine ligaments, leaving the uterus only attached by the connective tissue in the base of the broad ligaments which carries the uterine vessels, and it will not descend. It may be pulled down under these circumstances, but it goes up again at once if the traction is taken off. When the parametric tissues, however, are divided with intact ligaments and pelvic floor, the uterus will descend and remain in a lower position.

Thus we see that clinically, at least, it has been long recognised that the real supporting structures of the uterus are the parametric connective tissues, or, in other words, the perivascular connective tissue of the uterine blood-vessels.

If we consider the anatomical relations of the structures above mentioned, we note that strictly the round ligament and ovarian ligament are one and the same structure, whose function is to draw down the uterus and ovary in fœtal life, as the gubernaculum draws down the testis in the male. These ligaments are therefore vestigial structures, and could not be expected to have a supporting function. The clinical fact that the round ligament varies enormously in size and muscularity, sometimes being hardly recognisable, bears out this view. The utero-sacral, broad and utero-vesical ligaments

are merely peritoneal folds, the broad ligament in particular acting solely as a meso-ovarian and a meso-salpinx. They therefore are not supporting structures.

Peter Thompson has shown that the muscle-layers of the pelvic floor consist of an upper and a lower layer. The upper layer forms the pelvic diaphragm and is represented by the levator ani; the lower layer forms the sphincters of the openings of the rectum and vagina.

The levator ani is strictly the representative of the tail-depressing and tail-wagging muscles in four-legged animals, and thus in man must be regarded as a vestigial structure. In proportion, however, as man has lost his tail, the levator ani has gained importance in its relations to the rectum, forming a sling around it and assisting as a sphincter to it and the vagina. Further, the levator ani forms the funnel-shaped pelvic diaphragm with sloping sides, and as such cannot have much effect in supporting the pelvic viscera.

Keith, Derry, Cameron and Thompson have recently contributed articles on the pelvic fascia, and show that its text-book ramifications, once the bugbear of us all, are purely artificial and should therefore be discarded. The pelvic fascia is merely the remains of the mesoblastic tissue in which the muscles are developed, and which ensheaths the viscera and blood-vessels. In places this sheath is enlarged and strengthened, and this is particularly the case around the vessels which supply the uterus, vagina, bladder and rectum. Cameron particularly describes the ensheathing connective tissue, and shows how it is firmly attached to the innominate bone and the obturator fascia. In the upper part of this sheath we find the obliterated hypogastric artery; below it covers in the anterior branches of the internal iliac artery and is continued on to the uterus, vagina and bladder. The rectum has a connective-tissue supporting layer of its own, arising from the middle line behind around its vessels; hence the rectal supports are quite distinct from those of the uterus, vagina and bladder. This explains why it is that the rectum so seldom descends with prolapse of the uterus, vagina and bladder. Thus it is clear that in the present state of our knowledge the movable portions of the pelvic floor are truly supported by the perivascular connective tissue around their proper blood-vessels, and owe very little to their ligaments or the pelvic floor for the maintenance of their position.

It must not, however, be supposed that the pelvic floor may not in some way contribute to descent of these organs if it is injured. Clearly by dividing the fibres of the two halves of the levator ani from their attachment to the perinæal body, the potential opening through the pelvic diaphragm is enlarged; hence if the uterus is loose it will descend more easily along with the bladder and vaginal walls. But we know that in many instances these organs do not descend in spite of severe perinæal laceration, hence we must conclude that these organs have not had their attachments previously loosened. How is this loosening of the attachments of the uterus, vagina, and bladder brought about, and what, then, is the real cause of prolapse? The real cause is stretching and want of tone in the perivascular fibrous tissues of all these organs. How is this stretching brought about?

First and above all as a result of labour. This has been long recognised, but not exactly how labour brought it about. Even now we do not know why some women have prolapse after labour and others do not, in spite of lacerations, but I think we can construct some theory upon the subject. Severe lacerations occur by far the most frequently in precipitate labours, where the first and second stages are short, the pains very powerful and the child perhaps small. These women do not necessarily get prolapsed viscera. I think this is because the cervix dilates rapidly, retracts over the head, and as a result the uterus remains at its normal height, and there is as a result no long-continued traction on its vessels and their sheaths. Supposing, however, the first stage is very long and complete dilatation is long delayed, does the head remain high up? Not of necessity; it may descend very low in the pelvis, and this must mean much stretching of the uterine vessels and sheaths, because the uterus is pulled down as the head descends. If this is long continued, the vascular sheaths must be overstretched, perhaps injured beyond recovery, and after involution the uterus remains loosely attached and so will descend easily. Premature application of the forceps through an incompletely dilated cervix will, by traction on the fœtal head, drag the uterus lower even than any natural process can, and so overstretching of vascular sheaths may be brought

about. It is reasonable to believe that even this treatment may not have a lasting effect in a healthy individual, but in an anæmic, weakly and ill-fed person all connective tissues are flabby and inelastic and so the results may be amplified. When the supports are loosened any increase of abdominal pressure brought about by straining in defæcation, chronic cough, or lifting heavy weights will necessarily cause the uterus, bladder and vagina to descend lower and lower.

Is there any other real cause of prolapse but labour? We must admit there is, but that it is very rare. We sometimes see complete procidentia in a virgin, and this is sometimes slowly, sometimes suddenly, produced. To explain these cases is difficult, but the patients are usually anæmic, ill-fed, and over-worked, and so have the conditions which predispose to inelastic extensible connective tissues, in addition to increased abdominal pressure from over-work.

The sequence of events in any case of prolapse is usually this: The first organ to descend is the bladder, pushing the anterior vaginal wall before it and so causing a cystocele. This puts traction on the cervix and retroverts the uterus. Traction continuing, the uterus descends and drags with it the posterior vaginal wall. This strips the posterior vaginal wall off the rectum, peritoneum descending to take a lower attachment to the rectum, and so the prolapse becomes complete. In a small percentage of cases, however, a rectocele precedes the uterine descent, or a rectocele and cystocele together. Rectocele is more difficult to explain, but it may be that the normal attachment of the vagina to the rectum is altered by cicatricial tissue from injury, and so the two cannot separate. It is supposed that perinæal rupture is the necessary antecedent of rectocele, because when the perinæum is deficient, during defæcation the fæcal mass is forced against the anterior rectal wall, and being less efficiently supported causes a bulge over the sphincter into the vagina. Frequent repetition of this ends in a permanent rectocele, with consequent traction on the cervix by the posterior vaginal wall.

It is interesting to notice here the effect of cystocele upon a uterus whose supports are *not* loosened. Here the uterus remains at its normal height in the pelvis, but the constant traction of a cystocele brings about the condition known as hypertrophic elongation of the cervix. The particular form of this is the one known as supra-vaginal hypertrophy. If the uterus is partially loosened and opposes some resistance to descent, we have hypertrophy of the cervix and gradual descent going on together, a condition not at all uncommon.

The symptoms of prolapse in itself are very indefinite and by no means pathognomonic. Many patients complain of "falling of the womb" who have it not at all. Pain is nearly always present, of a dragging character, and often bearing down, whilst sacralgia is the rule in all important uterine displacements. It is not, however, until some part of the genital tract appears at the vulva that the patient can give any real account of her feelings. The presence of a complete prolapse produces symptoms of its own, for the protruding mass is liable to injury by rubbing and excoriation, and so ulceration occurs, giving rise to discharge and often bleeding. It is curious that menorrhagia hardly ever accompanies prolapse, whilst it is quite common in backward displacements. This means that endometritis and uterine congestion are not caused by prolapse as they are by retroflexion. It is, however, in the bladder symptoms it causes that prolapse shows its only characteristic symptom, and that only in severe cases as a rule. Owing to the descent of the bladder and interference with the nervous mechanism of micturition the act becomes difficult. Sometimes it cannot be accomplished in the sitting posture, but only when the patient lies down and the bladder consequently goes back into its normal position. No doubt the muscle coats of the bladder become stretched and sometimes hypertrophied in addition as a result of these difficulties. Cystitis sometimes accompanies prolapse, but by no means as often as we might suppose. Owing to the fact that a portion of the bladder is below the level of the urethra, it sometimes happens that the act of micturition does not empty the bladder. Hence there is residual urine, and sooner or later this will become infected through the urethra and cystitis results. Then of course frequency of micturition occurs and the urine shows the usual characteristics. It must not be forgotten, however, that a mild degree of cystitis may occur with acid urine, with only sufficient pus to be recognisable by the microscope after centrifugalisation. All degrees of prolapse may occur without any effect on pregnancy, and it

is not at all uncommon to see a pregnant uterus with the cervix outside the vulva. In such cases there is no special tendency to abortion, because as a rule the endometrium is healthy. The results of uterine prolapse may be discussed according to the degree of descent and to the effect upon the muscles, fasciæ, and peritoneum of the pelvis. It is usual to speak of three degrees of prolapse, namely, the first, in which the uterus is retroverted and rather lower than usual, the second, where the cervix reaches the hymen, and the third, called procidentia, in which the vagina is everted and the uterus wholly or partially outside. As a rule the incidence of these three degrees is gradual and progressive, though it does not follow that a given case will ever acquire the third degree. In some cases, however, the incidence is *sudden*, and as a result of some severe strain the complete procidentia is produced all at once just as an inguinal hernia may be produced by a sudden effort. The latter rarely occurs in married women in my experience; they commonly have the slowly-increasing form. I have seen two cases in virgins in which the third degree was produced suddenly and the uterus remained outside.

The chief effect upon pelvic tissues is shown in the peritoneum, which progressively descends as the posterior vaginal wall strips off the rectum, and produces greater and greater depth to Douglas's pouch. This is an incurable condition apart from very severe operations, whilst the stretching of the pelvic connective tissues may be to some extent recoverable if treated in time.

Treatment must be discussed according to the time after the first onset that we see the cases, or according to the severity of the lesion. If prolapse occurs within a few weeks of a first confinement we may sometimes be able to effect a cure by immediate palliative means, because we have no permanent after-effects to deal with. If there has been a perinæal laceration which has not been repaired, this, as a matter of course, must be treated first. For although ruptured perinæum is not the prime cause of prolapse, it certainly predisposes to cystocele, and must therefore be treated. I cannot pass on from this without urging the necessity for the immediate repair of *all* perinæal lacerations. It is an absolute axiom that any perinæal tear which injures more than the fourchette should be immediately sutured. Such injuries *never* heal primarily if left alone, but always by granulation, and so a defect in the perinæum must result. If the perinæum is complete we have to decide whether the case shall be treated by pessaries or by other means. In some cases the answer to this query will be found in the symptoms. If the patient has sub-involution shown by prolonged lochial discharge, it is well to commence treatment without pessaries. The best plan to pursue is to keep the uterus in place by daily packing with cotton-wool soaked in glycerine, with one daily douche of alum, one drachm to the pint. Internally, ergot, in the form of the liquid extract in one-drachm doses three times a day, is the only reliable drug which has a good effect upon the sub-involuted uterus. As the uterus recovers under this treatment there is some hope that its natural supports will also recover their tone and a cure result. If there is no sub-involution a pessary will give the best results. The ring pessary is the only one of any use here, and that size should be chosen which is the *smallest* which will keep the uterus in place. It is a great mistake ever to use pessaries too large. The pessary should be removed as soon as ever the uterus shows signs of keeping in its place without artificial aid. The results of this treatment will depend largely on the mode of life of the patient. If she is in good circumstances and does not have to work hard a good result may be expected.

We do not often, however, get the opportunity of treating a case so soon after its onset, but usually see them when all the tissues have settled down in an abnormal condition, and the pelvic organs are permanently displaced. Our treatment in these cases will depend upon the severity of the lesion. Practically we have to deal with three types of cases: those in which a cystocele is the chief lesion, those with cystocele and more or less descent of the uterus, and finally those with complete procidentia. All these conditions *may* be treated by pessaries of one kind or another, and often have to be so treated. But it must not be forgotten that pessaries will *not* cure these lesions—in fact they will probably tend to get worse. Also the condition of the vulval outlet must not be overlooked, because if it is very relaxed no simple pessary will remain in position. Therefore we may put the pessary question in a few

sentences thus: if a pessary will remain in the vagina in spite of straining efforts, if a pessary will relieve all the patient's symptoms, if the patient does not object to a pessary, but refuses operative treatment, to let her wear a pessary is a matter of convenience, and the only way to make the patient comfortable. In spite of the obvious disadvantages of pessaries, many women continue to wear them for years. If a ring will stop in the vagina it is the best to use. Sometimes when a ring is forced out on straining, it may be retained by wearing a firm pad outside the vulva strapped to a belt outside the stays. If a ring will not stop in we have no choice but to use a Zwancke, a cup and stem with straps, or one of its modifications. The Zwancke should only be used for intelligent persons who can be taught to remove it every night. If left *in situ* for any length of time it causes ulceration and may cause vesico-vaginal fistula. The cup and stem or its modifications often prove unsatisfactory if suspended by straps to a belt round the waist. Where this is so, some people find they can wear these pessaries if suspended by straps over the shoulders. I purposely do not spend much time discussing pessaries, for they are after all only a convenience and not a scientific means of treating these conditions. Further, it must not be forgotten that a uterus may descend almost to the vulva *without causing any symptoms*, and under these conditions no treatment at all is necessary. It is by no means necessary to use a pessary for a displaced uterus which causes no symptoms. I often have to deal with patients who are wearing pessaries who are no better for the pessaries, and whose symptoms are not caused by the local pelvic condition. To turn for a moment to the operative treatment of prolapse, practically the procedure is the same whatever the severity of the lesion, only the amount to be carried out is more the greater the descent. The first principle in these operations is the repair of the pelvic floor, and with it plastic operations upon the vagina and uterus. Whatever other operations we may do, this first principle always must be attended to. For instance, ventro-fixation is sometimes a useful adjunct, *but by itself* is quite useless to keep a prolapsed uterus in position. Hysterectomy even is advocated for very severe cases, but even after this operation the vaginal walls and bladder will prolapse unless plastic operations are done as well. We must not overlook the great fact that the uterus itself has nothing to do with prolapse, and removal of it will not cure the general pelvic floor descent.

The great operative difficulty to face is this: at present we have no sure means of dealing with the peri-vascular sheaths at the sides of the pelvis, which are the real supports of the uterus and whose stretching and tonelessness is the real cause of descent. The ingenuity of surgeons, however, is being turned in this direction, and the operation devised by Hastings Tweedy is a step in the right direction, but requires prolonged experience of cases operated upon before we can pronounce judgment upon it. The principle of this operation is that of uniting the bases of the broad ligaments in front of the cervix after freeing them from the uterus and vagina.

Every case must be operated upon according to its merits, and the severity of the operations must depend upon the severity of the lesion.

The usual sequence of operations is this: first to amputate the cervix if it is elongated; secondly, to remove a large area of the anterior vaginal wall, pushing the bladder up and bringing the connective tissue and mucous membrane together below it; thirdly, to repair the perinæum and remove redundant posterior vaginal wall after separating the rectum. All these operations must be done if the case requires them, and they may be done at one sitting.

As it is the supra-vaginal cervix which is elongated, it is necessary to first separate the bladder from the uterus after incising the vaginal attachment all round. Then tie those branches of the uterine artery which supply that part of the cervix to be removed, and finally unite the vaginal wall to the cervical mucous membrane after a transverse amputation.

Anterior colporrhaphy is necessary if cystocele is present, but it must not be a mere removal of vaginal mucous membrane. The bladder must be freed by dissection, pushed up, and then the paravesical tissues brought together with the vaginal walls so as to form a firm basis upon which the bladder may rest.

The perinæum is best repaired by Lawson Tait's flap-splitting operation combined with posterior colporrhaphy if the posterior vaginal wall is redundant.

In women past the menopause these plastic operations may be supplemented by ventro-fixation, or the operation of fixing the fundus uteri by sutures to the abdominal wall after making a median incision. If it is thought advisable to perform ventro-fixation in women who are still capable of child bearing, the organ should be fixed to *peritoneum only*, and by sutures as low down as possible, so as to leave the fundus free to expand should pregnancy ensue.

October 19th, 1908.

A CLINICAL LECTURE

ON

A CASE OF FEMORAL ANEURYSM TREATED BY EXCISION OF THE SAC.*

By C. H. FAGGE, M.S.Lond., F.R.C.S.Eng.,
Assistant Surgeon to Guy's Hospital.

GENTLEMEN,—Before reading you the notes of this case I should like you to look at the patient for a few minutes, so that you may realise his present condition.

You will notice that in the upper part of the right thigh there is a long scar, which, beginning just above and internal to the anterior superior spine of the ilium, runs downwards and inwards to the centre of Poupart's ligament. It then curves downwards with an inward convexity, and extends beyond the apex of Scarpa's triangle. This scar was made at the operation done here, and you will see that it is entirely healed except for a small granulating area near its centre. The patient has walked down from the ward, and you will observe that there is no marked difference between his two legs. When examined quite closely a small amount of œdema may be detected over the right shin, and the superficial veins of the right leg are somewhat dilated, but the venous obstruction on the right side is at the present time insufficient to produce any discoloration of the foot.

He will now return to the ward, and I will read to you an abstract of the Surgical Ward Clerk's report of his case.

Frank W—, æt. 26 years, fitter, attended my surgical out-patients for a painful tumour in the right Scarpa's triangle. He said that four years previously, while engaged at his work, he was hit by a crowbar in the position of the swelling. A year ago he had rheumatic fever, but as far as can be judged there was no evidence of endocarditis at the time. There is no history of syphilis. Eight months ago he noticed a small lump in the upper part of the right thigh, which has gradually increased, and for the past three months he has experienced an aching pain in the position of the swelling, which remains localised to this spot, and does not extend into the limb below.

On examination in the Out-Patient Department the tumour, which was situated immediately below Poupart's ligament, measured about four inches transversely by four inches vertically, and was raised an inch and a half above the normal surface. On palpation expansile pulsation could be detected over the tumour, which could be considerably diminished by compression of the external iliac artery, and when this pressure was removed the abnormal pulsation returned after two heart-beats. The right external iliac artery was felt to be larger than that on the left side, and this enlargement extended as far as it could be traced upwards. There is no bruit to be heard over the tumour, and no thrill can be felt.

It was thought by some of those who examined him that the posterior tibial pulse was delayed and smaller on the right side than on the left side.

On the diagnosis of traumatic aneurysm of the common femoral artery the patient was admitted and anæsthetised for operation.

The right external iliac artery was exposed by an incision corresponding to the upper part of the above-mentioned scar, which incision closely followed the classical method of Sir Astley Cooper. When the artery had been defined it was seen, as was suspected on manipulation before the operation, that its last one and a half inches was dilated, while above this its diameter appeared normal. Three strands of No. 4 French catgut were then passed underneath it, and each tied separately with a surgeon's knot, care being taken to place them upon the healthy artery, so that the lowest one when tied was distant at least one and a half inches from Poupart's ligament. It was now found that pulsation in the aneurysm, though diminished, had not entirely ceased, so that it was considered that this procedure alone would not be sufficient to cure the aneurysm, and that an attempt must be made to deal more radically with the sac. The deep epigastric artery, which had been exposed at the inner end of the wound, was divided between ligatures, and the incision was continued downwards to the apex of Scarpa's triangle. At this spot the superficial femoral artery was exposed and divided between ligatures. In dissecting the flap of skin and subcutaneous structures off the aneurysmal sac with a Durham's elevator, its limits were clearly defined, and the anterior crural nerve, which was adherent for some three inches, was peeled off its surface. It was now found that the

* Delivered at Guy's Hospital.

superficial femoral vein, as it passed up behind the sac, was adherent to it, and so firm was this adhesion that at the point where it is joined by the deep femoral vein its wall became so thin that it seemed impossible to avoid opening it. Both the superficial and deep femoral veins were therefore isolated and ligatured, and after the deep femoral artery had been secured the sac was gradually dissected out from below. In doing this the common femoral vein was injured, but owing to previous ligature of its tributaries no serious hæmorrhage ensued. The external and internal circumflex vessels which arose from the sac also had to be ligatured, and finally the aneurysm was completely separated, the only other vessel needing ligature being the deep circumflex iliac artery. The common femoral vein was tied just below Poupart's ligament, and after the wound had been washed over with normal saline solution it was sutured throughout its whole length, except for a drainage-tube placed near the lower angle.

After removal the aneurysm measured $2\frac{1}{2}$ in. vertically, $3\frac{1}{4}$ in. antero-posteriorly, and $2\frac{1}{2}$ in. transversely; it was fusiform in shape, two thirds of its bulk projecting behind a line drawn through it to represent the line of the normal artery. The superficial and deep femoral, external and internal circumflex arteries arise from it; above the aneurysm the external iliac artery is dilated to twice its normal size for a distance of one inch.

For forty-eight hours after the operation the lower limb, which had been carefully elevated and kept warm with hot bottles and blankets, presented a very mottled appearance, but at no time did it become at all cold, though the patient said that he had no feeling in it. The deeper parts of the wound healed by first intention, but at the inner edge of the wound the skin became black and necrosed at the spot where I demonstrated to you that there is now a small granulating area. On the outer side of the right heel a blood blister of the size of half-a-crown was noticed some ten days after the operation, but it is now drying up, and has given rise to no further trouble. The patient soon regained sensation, and normal power has now returned in the leg. The pulse in the right popliteal artery has returned, but we are unable to detect the right posterior tibial pulse, though it is now nearly six weeks since the operation.

The diagnosis of aneurysm was in this case easy, for as I review the clinical signs suggestive of such a lesion you will observe that most of them were present in our patient.

(1) Tumour in the course of a main artery.

(2) Pulsation synchronous with the heart-beat and expansile in character.

(3) A systolic bruit heard over the tumour.

(4) The effect of compression on the artery above the aneurysm. This diminishes the size of the tumour and usually stops the pulsation, which, on relieving the compression, returns gradually, and regains its usual amplitude after two or three beats of the heart.

(5) The effect on the pulse of the arteries below the aneurysm. This is usually delayed, and the pulse is smaller than in the corresponding artery on the opposite side.

Besides these local signs in the neighbourhood of the aneurysm, diagnosis may be assisted by the occurrence of complications or by pressure effects exerted by the aneurysm upon the surrounding structures. Under the former heading must be placed:

(*a*) Interference of the nutrition of the toes or feet, sometimes ending in *gangrene*, which is due to the diminished blood supply of the peripheral parts, and is especially likely to occur if the aneurysm is sufficiently large to interfere with the accompanying vein, or if the artery below the aneurysm is blocked owing to detachment of portions of clot (embolism).

(*b*) Œdema or venous congestion of the limb, due to pressure upon the veins and the main lymphatic trunks.

(*c*) Compression of the nerves, giving rise to referred pain.

(*d*) Impairment of movement of an adjacent joint may be due to erosion of the bones forming it, by the aneurysm.

(*e*) Adjacent structures may be displaced, but this sign is only of importance in connection with aneurysms of the trunk.

Differential diagnosis.—The differential diagnosis, when the aneurysm contains fluid blood, must be made from:

(1) A tumour or an abscess situated over the artery, from which pulsation is transmitted to the tumour. Such a condition may usually be excluded by the fact that the pulsation is heaving and not expansile, by its size not being altered by com-

pression of the artery above, and by the fact that the pulsation returns in full force immediately digital compression of the artery above is relieved. Also it is unusual for the pulse in the vessels below to be much affected by such a condition.

(2) The artery may be pushed forwards or so displaced by an underlying tumour that pulsation is met with in an abnormal position. Here it should be noted that pulsation can only be detected in the line of the artery, and that it is absent over the rest of the underlying growth.

(3) Pulsating sarcoma may give rise to difficulty, but in this case pulsation is less forcible and regular, the consistency of the tumour is more variable, and it is usually ill-defined, owing to involvement of surrounding structures. Compression of the main artery will also but slightly diminish its size.

When an aneurysm is met with in which coagulation has taken place it must be distinguished from solid tumours in a similar position. Thus, in a case of my own a thrombosed popliteal aneurysm, giving rise to commencing gangrene of the foot, was mistaken for an enlarged semi-membranosus bursa.

When pain is the chief symptom complained of, and the aneurysm is not so large as to be detected as a tumour, it is important to bear in mind the possibility of its presence in order to avoid the mistake of confusing such a condition with rheumatism or sciatica.

Pathology.—I am unable, in the time at my disposal, to deal thoroughly with the antecedent conditions which give rise to aneurysmal dilatation of an artery, though in attempting to decide what was the pathology in this patient I shall briefly run through the common causes of such a condition. The history in this case at once suggests that traumatism was the exciting factor, and while they may be due to either single or repeated injuries, traumatic aneurysms are typically of the sacculated kind, whereas I think you will agree from inspection of my patient's aneurysm, which I have passed round, that the whole circumference of the vessel is diseased, so that it must be classified as of the fusiform variety.

You all know that the commonest agent in the production of aneurysm of the large vessels, such as the aorta, is a series of inflammatory and degenerative changes in the inner and middle coats of these vessels, known as atheroma; but I think this can be excluded by the patient's age and by the apparent healthiness of all his other palpable vessels.

A third cause is syphilitic endarteritis. There is here no history or other evidence that the patient has had this disease.

The fourth possible cause would be embolism or detachment of a portion of a thrombus from a diseased valve, which, becoming impacted in one of the peripheral vessels, gives rise to thrombosis and secondary degeneration of the arterial wall. Though the detection of this aneurysm closely followed an attack of rheumatic fever, there was nothing in several recent examinations of his heart to suggest that he has had any valvular disease, so that on the whole I think we must conclude that the local injury is the responsible agent alone, but, as I have pointed out, there are some difficulties to be overcome in arriving at this conclusion.

Treatment.—Operative treatment is usually available in dealing with aneurysms such as this, where one of the arteries of the limbs is involved, and provided that the disease is localised, and that the difficulty of avoiding adjacent important structures is not insuperable, extirpation of the sac is probably the ideal treatment. Professor Rose recorded in the 'Lancet' of 1883 a successful result after operation in a case very similar to my own. No doubt the maintenance of asepsis, and the avoidance of secondary hæmorrhage, which was an alarming obstacle to the surgeons who dealt with these cases before the Listerian period, is a most important factor, and has had to do with recent successes in this direction. But, as Pearce Gould has pointed out, such an operation, interfering with the circulation only in the immediate locality of the aneurysm, does not call for so free or extensive a collateral circulation as is necessitated by the Hunterian operation of ligature. At the same time extirpation is impossible in many situations where operative treatment is desirable, and in none is it more difficult than in dealing with aneurysms in the popliteal space—perhaps the most common situation—owing to the difficulty of avoiding injury to the adjacent vein and nerves.

An alternative procedure in the present instance would have been that known as the old operation of Antyllus (A.D. 300), in which the artery affected is secured above and below, the aneurysm itself

then freely incised, and after the clot has been evacuated healing is allowed to take place by granulation. The great disadvantage of this in the present instance, now that the risk of secondary hæmorrhage can be eliminated, would have been the long period of convalescence which would have been necessitated.

Ligature of the external iliac immediately above the aneurysm, after the method of Anel (A.D. 1710), could not have been carried out owing to the fact that the sac extended up to Poupart's ligament, so that ligation between the aneurysm and the deep epigastric and deep circumflex iliac arteries was impossible; as ligature of the external iliac artery higher up failed to entirely stop the blood flow through the aneurysm, it is clear that this method alone could not be relied upon to effect a cure (Hunter's operation, A.D. 1785).

In the 'Annals of Surgery,' 1903, Matas introduced a method of arteriorraphy, which I had hoped to make use of in this case. But when, at the very beginning of the operation, the external iliac artery was exposed and found diseased above the aneurysm, I was forced to the conclusion that the case was not suitable for this procedure. By this method, when dealing with a saccular aneurysm, the circulation is temporarily controlled above and, if necessary, also below the aneurysm, which is then freely incised in its long axis, and the orifice leading from the aneurysm into the artery is closed by one or more layers of sutures placed within the sac, each passing through the inner and middle coats of the vessel. By interrupted sutures the redundant sac of the aneurysm is now pleated upon itself, so that the cavity is obliterated with the result shown in my diagram on the board.

In dealing with fusiform aneurysms two alternatives may be employed. In the first, after similar precautions, the sac is laid open, and all the vessels leading into it are closed with sutures (endo-aneurysmorrhaphy); in the second method, which is a modification of the method adopted in dealing with saccular aneurysms, a rubber tube—serving as a guide for inserting the sutures, and removed before they are tied—is placed between the two main orifices of the fusiform sac, and the artery is so narrowed by a row of interrupted sutures as to form a new channel of the same calibre as the vessel above. These methods are probably not available for wide application, and though, owing to the fact that they restore the arterial channel, and therefore interfere but little with the circulation, their adoption is very fascinating, yet greater experience and records of a larger number of cases will alone show whether they have any certain practical application.

Perhaps the most important and suggestive detail in my case was excision of the common femoral vein at the same time as extirpation of the aneurysmal sac, which was in this case necessitated, in my opinion, by the intimate adhesion of the vein to the aneurysm sac, for up to quite recent years such interference with both the main artery and vein of the limb was generally regarded as a certain indication for amputation, owing to the probability of resulting gangrene. That it cannot be held so now is almost entirely due to the adoption of antiseptic methods, by which the possibility of all gangrene is largely diminished. And that the above is no isolated instance is confirmed by a second case now in one of my beds, in which, in the course of an operation for recurrent sarcoma both the superficial femoral artery and vein were excised throughout the length of Hunter's canal, with no resulting harm to the patient's leg.

October 19th, 1908.

We have received from Burroughs Wellcome & Co. specimens of their "Tabloid" Capsule Calcium Iodo-Ricinoleate, gr. 3 (0·194 gm.). Calcium iodo-ricinoleate is a new salt containing a large proportion of iodine, which combines the therapeutic value of calcium and of the iodides. It is tasteless and odourless and is not affected by the gastric juice. It produces no digestive disturbance, and is well tolerated by patients who cannot take potassium or sodium iodide. Calcium iodo-ricinoleate possesses in a marked degree the valuable action of its components, and in syphilitic cases the results obtained compare favourably with those of treatment by the iodides. It has proved very successful in condylomata and other specific manifestations, whilst in stubborn cases of ulcers, including rodent ulcer, which resisted other treatment, it has been reported to produce a healthy granulating surface. Various affections of lymphatic glands and of the thyroid have been favourably influenced by the administration. Calcium iodo-ricinoleate is suitably presented for therapeutic purposes in the "tabloid" capsule, which undoubtedly offers the most convenient and satisfactory means of administration.

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A CLINICAL LECTURE

ON

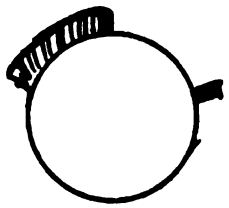
TRANSIENT HEART-MURMURS.

Delivered at St. Bartholomew's Hospital.

By SAMUEL WEST, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—All murmurs of the heart are not permanent, and to-day I propose to speak to you about some of those which disappear. Some have been called dilatation-murmurs, and I shall have to speak of these too.

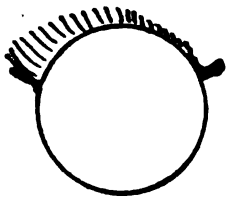
It is always well to agree as to what we mean by a certain term before we begin to discuss it, so we will begin with a definition. The ordinary definition of a murmur is very misleading. In order to make the matter clear I will give you a diagram



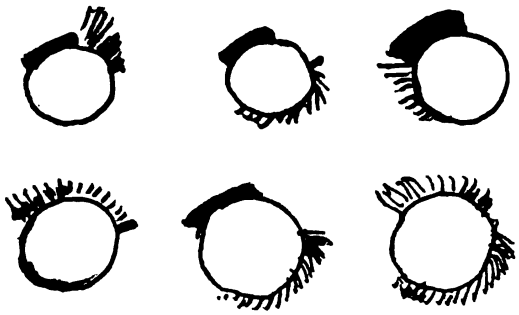
illustrating heart-sounds and intervals; and that brings me to the first important point. The sharpest distinction must be drawn between heart-sounds and murmurs. Murmurs are not altered heart-sounds. Murmurs may be present without heart-sounds, or sounds without murmurs, or any combination of both. What then is a murmur? The essence of a murmur is something which is heard between the heart-sounds, *i.e.* at a time when nothing at all is heard in health. Another point upon which it is essential that we should agree is the meaning of the word "systolic." You will find that on this point physiology books are not usually as explicit as they ought to be, and not so clear as clinical teaching must be if you are really to understand what murmurs are. You find

the adjective "systolic" used in varying senses. Some of your physiology text-books call the first sound the systolic sound, because it marks the commencement of systole; and the second sound the diastolic sound, because it marks the commencement of diastole. If with this use of the term "systolic" you speak of a post-systolic murmur you would mean something which came after the first sound and before the second, that is, between the first sound and the second sound. But that is the time when the ventricle is contracting, and that is systolic in the other sense, as occurring during systole. Yet a murmur cannot be at the same time systolic and post-systolic; that is absurd. The term "systolic" should be applied to what the ventricle is doing, and to nothing else. The ventricle is contracting during one period and dilating during the other, and anything which happens while the ventricle is contracting is called systolic in medicine; anything which happens while the ventricle is dilating is called diastolic; and the murmurs which are heard during the one period are called systolic, and during the other diastolic. Thus the two cardinal facts which you must ever bear in mind in the examination of the heart are: first, that you must distinguish sharply between heart-sounds and murmurs, and, secondly, when you use the terms "systolic" and "diastolic" you must apply those terms to what the ventricle only is doing, *i. e.* whether it is contracting or dilating.

What will strike you at once is that the diagram I have drawn does not agree with that given in most physiology text-books. What you get there is a drawing something like this, and the first



sound is depicted as shading off like the tail of a comet. I do not believe anybody ever heard all that; I cannot hear it. There is no means of proving what anybody does or does not hear over the heart, for the heart cannot yet write its own record. Murmurs are often described as replacing heart-sounds. That is not the case with diastolic murmurs at all, for the second sound is too short. A murmur may occur after the second sound, but it cannot merely replace it. When you come to the bedside you soon learn that you are entitled to divide the systole into two halves, one of which corresponds with the sound and the other with the interval, and in that interval you may hear something abnormal. That abnormal sound is the essence of a murmur; so that the definition of a murmur would be *an abnormal noise heard between the heart-sounds when nothing ought to be heard in health.* That is rather long, and it is better conveyed by a diagram. It is most important to distinguish between heart-sounds and murmurs, and that can be easily done upon a diagram.



What is the cause of a murmur? A murmur is caused by what has been called a fluid vein or jet, or eddies if you like, produced by some disturbance in the stream of blood. So long as the blood is flowing quietly through a tube no sound may be produced, but if an obstruction be put into it and the blood has to get past that obstruction, a fluid vein or jet with its accompanying eddies will be produced, and become audible.

The noise thus produced will depend on the force of the stream. And therefore you might suppose that murmurs might come and go, and this often happens. Some of them depend entirely on the force with which the heart is contracting, so that even a murmur due to permanent organic disease might not be audible in all states of the circulation. You might find in a patient who had just come into the hospital no murmurs which you could detect, but yet in a little while you might be surprised to find a distinct murmur audible. You might think at first that you had overlooked it. Perhaps you might have done, but it is quite likely that it was not there, for the patient being very ill when admitted, the circulation may have been too feeble, but as the patient improved the circulation

became more forcible and the murmur became audible.

The next point to determine is where these murmurs are loudest and in what direction they are propagated. They will follow the direction which the streams or little jets take when projected into the vessels. Let us take the aorta. If I fold my left hand and place it on my chest over the præcordium, sticking out my thumb, my thumb represents the aorta, which runs up towards the right clavicular joint. If a line be drawn straight down through my thumb to the apex of the heart, that gives you what I have called the *aortic axis*, and it is up and down that line that aortic murmurs are propagated; systolic upwards towards the right sterno-clavicular joint, diastolic downwards towards the apex. It is on the left side, in the third intercostal space, that the left ventricle is nearest to the surface of the chest, and where it is not covered by the other parts of the heart. And it often happens that this is the very spot where an aortic diastolic murmur may be heard and nowhere else. But generally speaking, aortic murmurs are propagated up and down that line, which you may call the "aortic axis."

Now, as to the mitral murmurs, that is to say the murmurs produced at the mitral valve. The left ventricle is in front, and the left auricle behind. If a skewer were taken and thrust in at the apex of the heart, and brought out a little below the angle of the scapula in the inter-scapular space, that skewer might, with luck, pass through the mitral orifice. This may be called the *mitral axis*. There are only two places where you can get near the axis from outside, one in front and the other behind. When the blood is passing from the auricle into the ventricle it is passing from the back to the front, and consequently that murmur will be heard in front, and very likely not behind at all. When the murmur, like an ordinary regurgitant murmur, is passing from the front to the back, the jet of blood is going to the back, and therefore we shall hear it not only in front, but also behind. That is how organic disease of the mitral orifice is diagnosed. I want to lay a little stress on these points, because this is the normal or the rule for the ordinary murmurs of organic disease. The murmurs which I shall speak of directly do not follow these general rules.

One remark before going further, as to the value of the heart-sounds in disease. It is most important when a patient has heart disease to examine the heart-sounds carefully, for this may be laid down as a general principle: that those lesions are least serious in all probability, where the heart-sounds are still audible, and those are the most serious where the heart-sounds are most altered or not audible any more. The loudest murmur does not necessarily indicate most disease or the worst lesion. If you put your finger over the outlet of a tap and try to close the tap with your thumb, you get a little spurt of water at great force, and probably at the same time it makes a good deal of noise. Take your finger away and the water flows out without much perturbation, and with very little noise. That is what often happens in the case of the heart. A small pin-point hole will cause a little jet of blood to be projected with very great violence into the stream, making much disturbance and many eddies, whereas in a much larger amount of disease, where perhaps the whole of the aortic valves are diseased, or destroyed, there may be a comparatively low pressure and little noise. Therefore it is very important to bear in mind that the loudest murmurs do not necessarily mean the gravest lesions.

Now, we may **classify our murmurs into two groups according as they depend on disease of the valves or not.** A large number of murmurs of the heart depend on valvular disease, and I have referred to some ways in which the diagnosis of the seat of disease may be arrived at. In the second group there is no valvular disease as far as we know. It is to this group that most of the transient murmurs belong, for as they are not connected with permanent organic disease, with the disappearance of the transient causes the murmurs will pass away. Of these transient murmurs some are heard over the aortic area, some over the pulmonary, some over the mitral areas, and some over the whole præcordium; and lastly there is the important group of what are called dilatation murmurs—*i. e.* murmurs associated with, and in some way caused by, dilatation of the heart.

All systolic aortic murmurs do not necessarily mean obstructive disease of the valves. **Transient aortic systolic murmurs** are not at all uncommon in candidates for life insurance. Possibly the applicant is a nervous individual, frequently a young man who is going to get married soon and

is required to insure his life, and he comes feeling in a general state of trepidation and excitement, and then you will very likely find quite a loud systolic murmur just above the aortic valves. Many cases have been rejected on that account. I remember a young man who came to have his life insured under these circumstances and the examining doctor put his ear to the chest and said: "Young man, walk through life; good morning," and rejected him. The young man had no disease of his aortic valves, was accepted by other offices as a first class life, lived an active life and died an old man.

I have no doubt it was one of those murmurs which come from excitement, and which pass away. In such a case if the candidate sits down quietly and his attention be drawn to other things, in a short time, perhaps a few minutes, the murmur will be found to have disappeared; or if not, he may come for examination another day when the idea of examination has become more familiar to him.

One of the general conditions under which a murmur is produced occurs when the blood passes from a narrow into a dilated space.

The conditions would be the same if there were dilatation at one part of a vessel while the orifice leading to it was normal, as if the orifice were narrowed and the rest of the vessel normal. Why do I mention that in this connection? Because I believe that it is the explanation of the murmurs we are speaking of, which occur under conditions of excitement in vessels. Excitement leads to a temporary dilatation of the aorta with a consequent murmur, which disappears so soon as the dilatation passes off. Vascular dilatations under excitement are common enough. Thus, if you look fixedly at a nervous person you may easily produce a blush, and oddly enough, very likely on the very part of the body you are looking at. If you are looking at the face the blush will be on the face, but it may be on the chest or abdomen, or whatever part you are to examine. These **nervous dilatations** are not limited to small vessels. There are curious conditions of big vessels of an exactly similar nature, which come and go, in which portions of the vessel may dilate and even exhibit a murmur; you can see or feel it dilated and pulsating, you can hear the murmur, and yet there is no disease at all, and soon all the signs disappear. This has long been familiar as it occurs in the aorta, and I daresay all of you have seen "pulsating aortas." In a patient with pulsating aorta there may be other pulsating vessels too. Sometimes you can see them all over the body, sometimes you see them in other parts and not in the aorta. I have seen such a condition in the carotid and in the subclavian, and I have more than once seen such cases diagnosed as aneurysm. Yet it is merely a transitory dilatation. These cases were originally described very many years ago by Laennec.

To the class of case I am speaking of the term "mimic aneurysms" was applied by Sir James Paget. It has rather dropped out of the literature recently, and I think you will hardly find anything about the subject in the ordinary text-books.

Now let us consider what we may call the **hæmic or venous murmurs.** Those are transitory, as you know: they are not always there. They vary with the position of the patient; they come and go according to the force of the circulation, the excitement of the patient, and so on. What is the explanation of them? Probably you find hæmic murmurs often described as due to a watery condition of the blood. It is true that in chlorosis the blood is pale and that it contains very little hæmoglobin and the specific gravity is low, but after all there is no proof that that is the cause of the murmur. I believe that these hæmic murmurs are in most cases due to exactly the condition of things which I have described here; the patient is anæmic, and in consequence the tone of the vessels is low, and therefore the vessel dilates. But when a vessel passes through a stiff fascia it cannot dilate there, but it does above and below it, and in that way the conditions for a murmur are fulfilled. And if the vessels are weak and offer very little resistance the posture of the patient may make a great deal of difference: veins might dilate in one position but not in another. The hæmic murmurs in chlorosis are often heard when the patient stands up and are not audible when the patient lies down, and that is a good illustration of what I mean. I think it is the explanation of the majority of these cases, because the murmurs are heard at points where the veins pass through fascia. Perhaps that explains the pulmonary murmur too, for in people who have what are called anæmic or hæmic murmurs the

pulmonary valves themselves will not stretch, but the vessel itself may dilate, and thus the requisite conditions are produced.

There are one or two odd murmurs which come and go **in connection with chorea.** You know a patient with chorea is very liable to have some valvular disease of the heart, and there is a close relationship between chorea and rheumatic fever. Many people say they are the same disease. Rheumatic fever is the common cause of acute endocarditis, and many cases of chorea get endocarditis too. Rheumatic fever may follow chorea, or chorea rheumatic fever. So without saying they are the same thing we may at all events say truly that they are closely related. Personally, I do not think they are the same disease. Measles and whooping-cough are not the same thing, but they stand in some relationship and one often follows the other; and I think that is the sort of relationship which exists between chorea and rheumatic fever. The reason I mention these two subjects I will tell you directly. Why do murmurs come and go in chorea in the curious way they do? Some of them no doubt depend on the fact that the heart is weak and dilated, and therefore the murmurs belong to the dilatation class. But not all of them belong to this group. While you are listening to the heart-murmurs you will hear every now and then a distinct period in which there is no murmur at all, and I think the explanation of that probably is that the heart is beating with different force at different times, for the heart's action may be irregular not only in rate and rhythm but also in force.

Transient murmurs occur **during chorea and rheumatic fever.** You hear them one day and then they go, or you hear them for a few days and cannot hear them afterwards. These have been described as curable murmurs, or as murmurs of cured heart disease. The explanation suggested is that there had been little vegetations on the valves, sufficient to throw the blood into vibration and produce the murmur, and that these were not permanent but gradually disappeared as the patient got better. That may be so, but so far as we can judge without actually seeing inside the patient's heart vegetations do not often disappear in that way. If a patient dies, vegetations are often found which were not expected to be present, and I do not know how it can be proved that they have been present and disappeared. The murmurs disappear, but I think that depends upon other causes, for instance, on the force of the heart's contraction, and on the degree of dilatation of the cavities.

This brings me to the subject of **dilatation murmurs.** To diagnose these there must be first of all evidence that there is dilatation, and that of course we obtain by means of the area of cardiac dulness. The cardiac area must be larger than normal. Of course you can diagnose by the shape of the cardiac dulness what part of the heart is dilated. We may say the right auricle or the right ventricle is dilated when the cardiac dulness extends well to the right. We may say the left ventricle is dilated when the apex is pushed downwards and outwards. We may say the left auricle is dilated when the dulness is square-shaped and extends upwards to the left. It is very important to be quite sure what parts of the heart are dilated. I described to you last time I lectured a very curious and interesting case in which there was a very odd murmur indeed which did not at all fit in with the ordinary valvular lesions of the heart. The conclusion which we arrived at from the character of the murmur and the shape of the cardiac dulness was that it was a murmur produced, not at the mitral valve, but in the left auricle itself. And when the patient died a large mass of vegetations was found just where we supposed they had been. You must associate together the dilatation as ascertained by the cardiac dulness with the murmur and its character before you are justified in diagnosing a dilatation murmur.

What, as a rule, are the characters of dilatation-murmurs? First of all, they are almost always systolic. Occasionally they are diastolic, and sometimes they are continuous. But speaking generally these dilatation-murmurs are systolic, they are soft and blowing in character, not harsh and rasping. The heart-sounds are not materially altered; they may be weak, but they are present. And lastly these dilatation-murmurs are more or less localised, that is to say, localised to the parts where dilatation has occurred, wherever that may be. And they are not propagated in the direction in which they ought to be if they were due to valvular lesions, that is to say, in the directions I have described, *i. e.* along the aortic or mitral axis.

I said at the commencement of the lecture that murmurs must be due to eddies. How are eddies

produced in a dilated heart? that is to say, in the heart itself. It is much more easy to explain why murmurs occur than it is to explain why they do not, and I think it is one of the most wonderful instances of physiological adaptation that the heart's action and the circulation should go on so smoothly and gently that in health there should be no sound or disturbance produced. Long inheritance and the gradual development and adjustment of the heart to the needs of the circulation and the condition of the vessels have resulted in the carrying on of the circulation with the minimum of friction, because eddies mean friction, and friction means energy not properly utilised. Anything which disturbs the normal relations, such as dilatation, may cause eddies and possibly murmurs. You will find it stated very dogmatically in some books that these murmurs are not due to dilatation. I have called them dilatation-murmurs intentionally, because I want to raise the question and to discuss it. Ludwig and Hasse stated that the valves of the heart are not in health completely competent to close the orifices of the heart unless by means of the muscular contraction the orifice is first narrowed. That statement rests upon certain physiological experiments of a very crude character, which, so far as I know, have never been confirmed, and against which there is the strongest possible clinical evidence. Now let us see what that statement amounts to. The heart, I suppose, from the point of view of life, is the most important organ in the body. You may destroy half or even three quarters of the kidney, and the patient will go on living. You may destroy three fourths of the liver, or three fourths of the lung, even three fourths of the brain or the whole brain sometimes, and yet the patient will go on living. But that would not apply to the heart if the statement made by Ludwig and Hasse were true. Is it likely, one may fairly ask, that the heart should have such a narrow margin between health and disease as is indicated by that theory? Because the heart is a pump, and a pump in which the valves leak is not a good pump. If the valves of the heart leak even a little, that heart is not doing its work properly and becomes defective. Is such a theory as I have mentioned probable?

An *à priori* argument is a very dangerous one, because it implies theoretical conceptions of your own which may not be correct, but we will go to clinical evidence to disprove the statement. What is that evidence? We will consider first the case of a dilated left ventricle and left auricle. A systolic murmur, blowing in character, is heard over the ventricles but nowhere else. We are told that is due to regurgitation through the mitral valves because, according to the theory referred to, the heart is weak and dilated and the valves do not close the orifice. If that were so and the blood passed back through that orifice because the valves did not act, the murmur ought not only to be heard in front, but it should be propagated to the back and heard behind, as the murmur due to mitral organic disease is. That, however, does not occur. Why? Because there is no regurgitation. Every now and then this dilatation murmur is propagated, as it ought to be, with organic disease, and then regurgitation must occur. I saw a case some time ago where a patient came into the hospital with extreme dyspnœa and bronchitis, deeply cyanosed, and very ill. He was taken into the hospital at once. He had a loud apex murmur, heard in front and behind. We therefore concluded that it was a case of mitral regurgitation with secondary bronchitis. But to our surprise, as the patient got better the murmur disappeared, the heart returned to its normal size, and he went out well. Twelve months later he came back with an exactly similar condition. For the second time also he got quite well and went out again. A third time he came back with the condition, and this time he died. I made the post-mortem examination and examined his heart carefully; the valves were perfectly healthy, but the orifices stretched and the heart itself was enormously dilated. In this case the murmurs were propagated in the right direction, and were heard in front and behind When dilatation has gone beyond a certain point the orifices may be stretched and regurgitation occur. But that does not so often happen. I have seen one or two other cases like this, but only these few out of an enormous number of dilatation cases.

If dilatation murmurs, then, are in most cases not propagated as they ought to be if they were due to regurgitation, we are necessarily driven to the conclusion that these murmurs cannot be due to regurgitation, and that the theory upon which this explanation is based is wrong.

Tricuspid regurgitation is a diagnosis frequently made: upon what facts is that diagnosis based?

A tricuspid murmur is produced by regurgitation through the tricuspid valve. Dilatation of the right side of the heart is extremely common, yet you do not often hear a murmur at all as the result, and when you do it is a soft blowing systolic murmur, heard over the whole area of cardiac dulness of the right side. How ought that murmur to be propagated if it were produced by regurgitation through the tricuspid valve? It should be heard over the lower part of the sternum and be propagated loudly on to the right side, along the fifth space, but in the ordinary cases of dilated right heart this does not occur. When there is organic tricuspid disease then the murmur is heard and propagated as it ought to be. Organic tricuspid disease is rare, but I have more than once diagnosed it on these grounds and found it when the patient died.

If, then, the ordinary dilatation murmur is not propagated in the same way as murmurs are which we know to be due to regurgitation, the theory of regurgitation cannot be accepted as explaining them. What I believe to be the true explanation I have already indicated, viz. that these murmurs are due to eddies set up within the dilated cavity itself.

October 26th, 1908.

THE exhibit of Burroughs Wellcome & Co. at the London Medical Exhibition included the results of some of the most recent advances in specialised research. Of particular importance were the arylarsonates, which unquestionably mark a new era in the treatment of protozoal diseases. These organic arsenical preparations may be administered in much larger doses than the highly toxic inorganic arsenical salts, and are now being successfully employed in the treatment of syphilis, sleeping sickness, etc. Results of the most encouraging nature are reported in connection with the use of "Soamin" in syphilis. Another arylarsonate, issued under the name of "Orsudan," is under trial in the treatment of malaria. The value of "Ernutin" preparations is now widely recognised among medical men. They present the active therapeutic principle of ergot in a state of purity never previously approached, and have achieved distinction from the uniformity of their action, which is ensured by physiological standardisation.

THE TREATMENT OF GONORRHŒAL AFFECTIONS AND OF SOME FORMS OF ARTHRITIS OF UNCERTAIN ORIGIN.

By J. PORTER PARKINSON, M.D.,
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How widespread through the body the results of a gonorrhœal infection may be is now well recognised; it is known that by direct extension in the female metritis, salpingitis, ovaritis, and peritonitis may be caused; and in either sex cystitis, pyelitis, and nephritis. These results, however, as a rule come under the care of the gynæcologist or the surgeon, and will not be further alluded to, though it seems probable from analogy that the treatment, so successful in the cases to be described, would be of considerable value.

Systemic gonorrhœal infection is of two kinds, septicæmia and pyæmia, and arthritis; and as it was in the former that this treatment was first found to be successful, I will begin with this form.

Some of the following cases are taken from a paper read by Dr. Soltau Fenwick and myself before the Royal Medical and Chirurgical Society, in 1906:

A married woman, æt. 39 years, was admitted into the London Temperance Hospital at the end of March, 1903. She had suffered for nine days from severe pains in the right wrist and left knee, had become feverish, and for two days had been delirious. On admission she was delirious and very anæmic. The skin was hot and dry, the tongue thickly coated with a greyish-brown fur, the axillary temperature was 101·2° F., the pulse 102, and the respirations 28 per minute. A few patches of erythema were visible upon the forehead and sides of the neck. The right wrist was swollen, and the dorsum of the hand was reddened and pitted upon pressure. Both knee-joints were swollen, and contained a moderate quantity of fluid; there was puffiness of the right ankle, and œdema of the dorsum of the foot. There was no purpuric eruption. The lungs appeared normal. The impulse of the heart was in the normal position and was feeble, and a soft systolic murmur was heard over the mitral area and conducted outwards as far as the anterior axillary fold. The liver was not

enlarged, and the spleen could not be felt though its area of dulness was increased. There was a purulent discharge from the vagina, and the uterus was fixed to the left side, but no disease of the pelvic viscera could be detected. The urine withdrawn by a catheter had a specific gravity of 1014, and contained a trace of albumen. The retina and optic discs were healthy. Though the general condition of the patient seemed to indicate pyæmia rather than rheumatism, full doses of salicylate of soda were ordered, and a small quantity of the vaginal discharge was collected for examination. Two days later the temperature had fallen to 100° F., the pulse-rate was 98, and the respirations 28 per minute, but the general condition was worse, and both the urine and fæces were passed into the bed. The vaginal discharge was reported to contain numerous gonococci, so the salicylate mixture was omitted in favour of one containing quinine and iodide of potassium, and the local treatment for gonorrhœa was adopted. The condition remained about the same for a week; but on April 8th she became decidedly worse; the temperature had mounted to 103° F., the pulse was 120, and the respirations 40 to the minute. The tongue was dry, brown, and cracked, the features were shrivelled, and the complexion had assumed a sallow hue, the mitral murmur had increased in intensity, and a systolic bruit was present over the aortic area. Numerous rhonchi and moist râles were heard over the right lung, and there was a friction sound over the anterior part of the base of the left lung. On April 14th, six days later, the patient was practically moribund, and was apparently only kept alive by repeated injections of strychnine. She was completely unconscious, the mouth was foul, the lips covered with sordes, the pulse feeble, varying from 132 to 140 beats a minute; there were signs of œdema at the base of the right lung, and of a moderate effusion into the left pleural cavity. A little turbid fluid withdrawn from this showed a few gonococci enclosed in the pus cells. In this desperate condition 10 c.c. of the anti-streptococcus serum prepared by Burroughs Wellcome & Co. were injected into the rectum. The following day, to our surprise, the patient was still alive, and was able to swallow a little nourishment. The temperature had fallen a degree and a half, but the pulse-rate had not diminished. A second injection of the serum was given. At midday on the third day after the first injection, that is, after the lapse of sixty-six hours, the temperature had become subnormal, the pulse was 108, and the respirations 34 a minute. For the first time the patient was quite conscious, and both able to speak and to swallow liquid nourishment with ease. She had recovered the control of her sphincters, and a wonderful improvement had taken place in her facial expression. The next day the temperature rose to 100° F., and continued at this point for nearly a fortnight, but this appeared to depend solely upon the pleural effusion, as it gradually declined when this became absorbed, and in a way retarded the progress of convalescence. In all three doses of the serum were administered by the rectum within five days. The pain and stiffness of the affected joints disappeared in a few days along with the signs of swelling and effusion. There was much insomnia during convalescence, but this was the only trouble, and she was discharged from the hospital on July 1st. Just before discharge it was noted that the cardiac impulse was forcible, in the nipple line, the mitral murmur was still heard, though less loudly; the aortic bruit, however, had disappeared. There was deficient expansion of the lower half of the left chest, due doubtless to pleural adhesion, and the urine still contained a trace of albumen. The vaginal discharge had quite ceased.

Our second case was a girl, æt. 18 years, who was admitted into the hospital on October 9th, 1903, for what was supposed to be acute rheumatism. Except for anæmia she had previously been quite healthy. The illness commenced a week before admission, with pains in the body and limbs accompanied by fever. The pains were chiefly in the right knee, ankles, and wrists, the knee-joint containing a small quantity of fluid, while the other joints were swollen and extremely painful. The skin was dry, the tongue furred, and the temperature 100·5° F. The heart, lungs, and other organs of the body were apparently healthy. The patient was treated for acute rheumatism, but in spite of large doses of salicylate of soda and other remedies the condition of the joints grew steadily worse, and the temperature rose till at the end of eleven days it registered 103·2° F. Meanwhile it had been discovered that the girl was suffering from gonorrhœa of several weeks' duration, and numerous gonococci had been found in the

vaginal secretion. This was treated by local injections.

Eleven days after admission pleurisy developed at the base of the left chest, and a soft systolic murmur became audible over the mitral area of the heart. Large doses of iodide of potassium and quinine produced no effect upon the course of the disease; pleurisy developing at the right base on October 23rd, while a pericardial friction appeared the next day.

Small quantities of turbid serum withdrawn from the left pleura and from the right knee-joint were found to contain gonococci, and the urine showed one tenth of albumen.

From this time the general condition of the patient grew rapidly worse; the temperature continued to range between 103° and 104° F., the pulse-rate exceeded 130 beats per minute, and there was great pain in the affected joints. On November 3rd, when all other remedies had failed and the patient appeared to be sinking, 10 c.c. of the anti-streptococcus serum, prepared at the Wellcome Laboratories, were injected into the rectum, and the dose repeated each day for a fortnight. After the second injection the patient expressed herself as feeling much better, the pain and swelling of the joints rapidly subsided, the appetite and sleep returned, and in spite of the effusions into the pleuræ and pericardium the pulse-rate and the temperature steadily diminished, until, on the thirteenth day after the first injection, they had both regained their normal state.

The patient made an uninterrupted recovery, but when discharged from the hospital there was some evidence of enlargement of the left ventricle and a soft mitral bruit still audible at the apex of the heart.

These two cases illustrate sufficiently without further examples the rapid and excellent results obtained by the rectal injection of this anti-streptococcal serum in cases such as above. And I may add that since this treatment was first used at the hospital not a single case has ended fatally.

It should be noted that these are not cases of a secondary infection occurring in patients suffering from gonorrhœal urethritis, as the specific organism was in each instance recovered from exudations into the joints, pleura, etc. They show therefore that this serum has some effect either in inhibiting the growth of the gonococcus, or of neutralising the effects of its toxins. As soon as we had satisfied ourselves of this, we instituted a series of control experiments by means of simple enemata and rectal injections of anti-diphtheritic and of normal horse serum, but in no instance did the patients experience any relief of symptoms as the result of them, nor did the physical signs of the disease in any way improve until the anti-streptococcus serum was substituted for the other forms of injection. The anti-gonococcus serum also proved of no value in our hands. Again, Dr. Dowson, formerly the director of the Wellcome Laboratories at Herne Hill, found to his great surprise that while the gonococcus readily grew in a medium to which the anti-gonococcus serum had been added, it failed to grow in a similar medium to which this anti-streptococcus serum had been added. He also found that no other serum had this inhibitive effect.

Another variety of gonorrhœal infection is arthritis. This is most frequently a periarthritis, the inflammation extending along the sheaths of the neighbouring tendons; it usually affects several joints, and is specially characterised by its resistance to ordinary treatment, by its long duration, and by the adhesions and stiffness resulting, and its tendency to relapse. The following is an example of this form, showing the effect of the serum treatment.

A young married woman was admitted suffering from pain and swelling of the wrists, ankles, and knees. Two years previously she had had an attack of gonorrhœa, followed by severe "rheumatism," for which she was confined to bed for six weeks. She had also had two attacks of peritonitis. The present illness began a fortnight before admission with a purulent discharge from the vagina, and swelling of the wrists and left ankle. The temperature was 101° F., and the pulse-rate 88. There was a profuse vaginal discharge containing gonococci in abundance. The wrist-joints were swollen and tender, and a puffy swelling extended several inches up the dorsal surface of each forearm. Any movement of the wrist and fingers caused great pain. The left ankle and knee were similarly affected. The patient was treated by antiseptic vaginal douches, followed by swabbings with a 2 per cent. solution of protargol, and internally large doses of salicylate of sodium combined with quinine were given. Radiant heat was

used to the affected joints. The temperature fell to normal after eleven days' treatment, and the vaginal discharge diminished, but the condition of the joints remained unaffected, and the right knee and ankle became swollen and tender. After another fortnight's treatment with iodide of potassium, etc., there was no further improvement in the condition of the joints, though the temperature remained about normal. The treatment was now abandoned, and 10 c.c. of the polyvalent serum was injected into the rectum every other day for three doses. By the sixth day the pain had almost disappeared from the wrists and knees,. and the swelling of the joints had subsided completely, leaving only a certain amount of stiffness and tenderness on movement. In another week this had also subsided, and the patient was discharged cured a fortnight later.

This case, along with others described in former papers, were selected as types of those treated during the early or experimental stage of the work, and in them the usual methods of treatment were tried before resorting to the use of the serum. They are not, therefore, to be met by the argument that they were of that mild type in which rest in hospital and antiseptic urethral or vaginal douches are all that is necessary to effect a cure. Again, it will be seen that the effect of the serum is so prompt that it is impossible not to believe that the improvement is due to its use. In some of the cases the gonococcus has been found in the fluid removed from the joint, though for obvious reasons this was not attempted in all; but in everyone the specific organism was obtained from the vaginal or urethral pus.

There are some cases, however, especially in men where no urethral pus was present, or anyhow, not detected; the effect of the treatment was equally good in these, while in those in which such a discharge was present, it disappeared without local medication.

It is well known how variable are the clinical forms of this disease. Osler mentions seven varieties: The arthralgic, in which there is pain without redness or swelling; the polyarthritic, resembling acute rheumatism; acute arthritis, affecting a single joint; chronic hydarthrosis; a form affecting the bursæ and synovial membranes; a septicæmic variety; and finally, the painful heel of gonorrhœa. Some of these varieties seem more easily cured by the serum treatment than others, though all kinds are improved. The chronic hydarthrosis variety seems specially resistant, and I have usually resorted to strapping the joint as well as the use of the serum. I have recently given the serum in three doses with an interval of two days between each injection. The amount used for the first and last dose are 25 c.c. each, and for the intermediate one 10 c.c. The serum is put up in these amounts by Burroughs and Wellcome, and it should be made clear that the polyvalent anti-streptococcus serum is the one desired.

The fact that the gonococcus may remain latent in the body for considerable periods led me to try the effect of the serum in cases where arthritic troubles were present, with a history of gonorrhœa previously but not in recent years; in some cases the results were just as striking as in the more evidently gonorrhœal arthrites.

A striking example of such a case is the following:

A gentleman aged over 60 had suffered from gonorrhœa when about 23 years old, followed by what was considered to be "rheumatism" three years later, during which, however, the urethral discharge reappeared. The attack crippled him for some weeks; it was followed by stiffness of the joints, and this had increased considerably during the last ten years. It frequently necessitated him mounting stairs on his hands and knees, and caused pain and difficulty in getting up from a low chair. The joints were not obviously enlarged or the ends of the bones thickened. He was treated by three rectal injections of the serum on alternate days, with the result that the pain and stiffness gradually improved, and after a couple of months he was able to walk and use his limbs freely and without pain. The improvement which began in this way three years ago has lasted up to the present time, and he describes himself as being better than he has been for many years. In a letter to me recently he states: "I am quite free from any symptom of rheumatism."

In some cases in which the serum treatment is successful there is neither a history suggesting gonorrhœa nor any evidence of its presence.

For example, a married lady, æt. 38 years, consulted me towards the end of July last year for attacks of "rheumatism," to which she had been

subject for about ten years. She had enjoyed good health, having had no illnesses except scarlet fever at the age of 23. She had never had acute rheumatism, but for ten years had attacks coming on without warning of severe pains in the joints, especially of the fingers and shoulders, with stiffness. The attacks lasted about two or three days, and then subsided, leaving the affected joints more and more stiff with each attack. On examination she had a healthy appearance. The heart and lungs were normal. The teeth had all been removed. There was some leucorrhœa. The affected joints were not deformed but more or less stiff, and there was some creaking on movement, while one or two of the interphalangeal joints were slightly fusiform from periarticular thickening. I looked upon the case as one of rheumatoid arthritis, and without much hope of doing good I injected three doses of the serum, and asked the patient to see me again in three months' time. The end of October she told me that in spite of the damp weather which had previously affected her she had been quite well for over a month, having had no attacks of pain in the joints. The leucorrhœa also was much less, though some discharge still remained. She had increased in weight. Some months later I heard again from her that she was still free from any joint trouble.

Another patient, the condition of whose joints resembled rheumatoid arthritis, was an unmarried lady who came to me from the midlands for "chronic rheumatism." Her age was 44 years, and there was nothing of importance in her family history till 1891, when she had an abscess in her mouth with decayed bone, and since that time she had never been strong. Since 1898 she has been nursing her mother, who is a chronic invalid. She has always had leucorrhœa more or less badly. For nine years she has suffered from pain and stiffness in many joints, those of the hands, ankles, knees, and hips being specially affected. There have been occasional exacerbations with more pain and swelling of the joints, fever, etc. There was some deformity of the phalangeal and metacarpo-phalangeal joints, resembling that seen in rheumatoid arthritis. I was unable to examine this case bacteriologically, but it appeared to be one of toxic arthritis, probably initiated by the previous bone necrosis, and perhaps having some relation to the leucorrhœa. She was given three injections of the serum, but there was no immediate improvement. Six months later, however, I had a letter from her in which she stated that for three months her joints had been much better, and the leucorrhœa had almost ceased. There had been no change in her mode of life, and no other treatment since the rectal injections. I saw her about a month ago, and though the deformity of the joints remains the same and their movement is impaired, the pains from which she had previously suffered and the acute exacerbations have disappeared for a longer interval than has ever before been the case.

This is one of those cases which are usually designated "rheumatoid arthritis," the pathogenesis of which is a matter of doubt, some considering it a morbid entity, while others consider the symptoms and morbid appearances may be produced by different kinds of bacteria and their toxins. It is the latter view that I think will ultimately be found to be correct. It is then not unreasonable to suppose, therefore, that a polyvalent serum such as the one used, which is prepared from forty-three different strains of streptococcus taken from different clinical conditions, such as septicæmia, ulcerative endocarditis, puerperal fever, acute rheumatism, etc., may at least be occasionally serviceable in some of these joint diseases of uncertain pathology. No bad effects follow the rectal injections, though an antitoxin rash is occasionally seen.

I have tried the remedy in many other cases similar to the one last quoted, the results being sometimes good and sometimes no effect being obtained, and up to now I have not arrived at any very definite indications as to its value in any particular case of rheumatoid arthritis, but it is at least worthy of trial where other means have failed.

In a fair number of cases of fibrositis and lumbago the treatment has been successful, but it is difficult to be quite sure in conditions like these, which are sometimes very fugitive and may disappear spontaneously, and also when, as in hospital patients, they may be lost sight of after one or two attendances.

October 26th, 1908.

We have received from Messrs. J. & A. Churchill a copy of 'A Dictionary of Medical Treatment' for students and junior practitioners, by Dr. Arthur Latham, for review.

THE MANAGEMENT OF SQUINT.*

By A. A. BRADBURNE, F.R.C.S.Edin.,
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THE term "squint" is misunderstood perhaps more than any other expression. A squint is simply a term used to express one of the visible evidences of a certain condition. Squint is not a disease, it is only one of the symptoms of, it may be, two totally different affections. Thus squint may be present as a result of defect in vision, as the result of a nerve lesion or muscular palsy, or as a reflex symptom of severe constitutional disturbance. Under such circumstances to name a disease by one, and not the chief, of its symptoms is both misleading and erroneous. Such unscientific procedure not only is evident in its nomenclature but is associated with the methods most commonly employed for its rectification.

The term "squint" then simply means a visible deviation from the normal position of the eyes, and forms but one symptom of a condition which may arise from various causes.

There are two chief varieties of squint, viz., paralytic and concomitant. In concomitant squint the eye which squints can be moved freely in all directions, whereas in the paralytic form the eye is limited in its movement in the direction in which its paralysed muscle acts. This feature of limitation of movement easily differentiates the two kinds and separates them into two totally different affections. The one I wish to speak about to-night is the former, viz.:

Concomitant strabismus.—This name not only indicates the presence of a deviation of the optical axes but also the following, viz.:

(1) A difference in the refraction of the two eyes.

(2) A deficiency in what is called the "fusion faculty."

And in addition there is nearly always found:

(3) A defect in the vision of one eye amounting to loss of all useful vision.

(4) An inability to bring the deviating eye to bear directly on any object.

It is evident, then, that the presence of concomitant strabismus indicates a complex state of matters, the rectification of which requires scientific handling. Not only has the outward visible defect to be corrected, but also those shortcomings associated with the vision. Thanks mainly to the teaching of Worth the treatment of squint has been completely revolutionised and put on a true scientific basis. For many years the only treatment adopted was that of haphazard operative interference, all the other factors being absolutely unrecognised, untreated, and ignored. The period of promiscuous operating with its train of dire results produced a natural reaction which resulted in the no less harmful advice being given to parents to wait until their child had outgrown the deformity. This line of treatment is no better than the older operative measures; one might just as well expect a rickety or tubercular child to outgrow spinal or tibial curvatures by an untreated course of waiting. It is a deep-seated belief and a grave error to believe that any case of true squint can be grown out of if left alone. There are many cases in which a squint does disappear, but such is never the case in concomitant strabismus; a squint which disappears belongs to the class of orbital imbalances known as "heterophorias," and in the commonly accepted interpretation of the word are not squints at all. If need be they can easily be distinguished by the presence of the fusion faculty, which, as has been pointed out, is always absent or deficient in a true case of strabismus.

The correction and management of a case of concomitant strabismus lies quite as much in the hands of the family physician as in those of the ophthalmic surgeon. Moreover, as the defect in vision sets in at an extremely early age, any delay in remedying and overcoming it will be productive of the most serious consequences. The fusion faculty on which so much depends for success in treatment cannot, except in rare cases, be awakened or developed after six years of age; the same applies to the recovery of vision in the deviating eye. Under such circumstances delay in commencing treatment means loss of the most valuable time, in fact in many cases means loss of all hopes of ever recovering either the fusion or visual faculty, and he who so advises takes upon himself a responsibility of the most momentous nature. Treatment, therefore, cannot be commenced at too early an age; by this, one does not mean that infants are to

* Delivered before the Medical Societies of Southport and of Chester.

be unnecessarily encumbered with spectacles, but when such are imperative eighteen months old is not any too soon to give glasses. Statistics prove that most squints begin before or about three years of age, and unless training exercises are commenced before six years old the chances of overcoming the defects in the eye and brain are very remote.

The technicalities connected with the types and varieties of strabismus would occupy too much time and space to enumerate here; such are fully dealt with in the text-books. For the same reason no further reference need be made to the theories of squint except to state that Donder's view that it arises from a disturbed relationship between the accommodation and convergence has been replaced by that of Worth, who considers it is due to a deficiency in the development of the fusion faculty. This defect is very frequently an inherited one. Breslau states that in 23 per cent. of his cases of convergent strabismus there was a history of squint in some relative.

The recognition of strabismus when present is not difficult. The two most reliable tests are the cover test and the mirror test.

The cover test.—This cannot be used in the case of very young children: in them the mirror test is the best. A piece of card or paper is taken and placed over one eye after the patient has been told to look at an object some distance away. If, then, the uncovered eye makes no movement we know that this is the fixing eye. Now with a quick lateral movement we cover the other eye, and if this moves we know that it was originally squinting. If the eye moves out it is an inward squint—a convergent strabismus; on the other hand, if it has moved in, the eye was originally squinting outwards—a divergent strabismus.

The mirror test.—This is the most satisfactory one to employ. The patient should be seated in a dark room with a lamp behind him. If an infant, it can be held on a nurse's knee who is seated as mentioned. A light is reflected from a retinoscopical or ophthalmoscopic mirror held in the hand at a distance of about two or three feet away. This will, when reflected into the eyes, immediately attract the attention of an infant, but an older patient will have to be told to look at the mirror. Reflected on the cornea of the eye will be seen a bright spot of light—the corneal reflex—and its position in relation to the pupil in each eye will afford evidence of any abnormality in the position of either eye. By interposing a card as a screen in front of the straight we note whether the deviating eye moves to look at the mirror; thus we learn in addition the presence or absence of what is called the power of central fixation. If the patient cannot fix the mirror with the deviating eye when the other is screened we know that the eye has become blind from non-use, a condition called amblyopia ex-anopsia. By covering up each eye separately and getting the patient to look in various directions we can ascertain what range of movements each eye possesses. This latter test readily differentiates paralytic squint from other types.

The treatment or management of a case of concomitant strabismus is as much the duty of the general practitioner as that of other human ailments. Just as in other diseases, expert advice can be sought if desired for guidance in any difficulties which may occur, but for the carrying out of the treatment itself no special knowledge of ophthalmology is essential. Successful management demands care, attention, and supervision for a long period of time, as it deals with the education of the visual organs of both the eye and the brain. For financial reasons alone very few can afford to receive this treatment at the hands of the specialist, and unless the family physician learns how to do it such cases will continue to remain uncorrected.

There are three main indications present in every case of squint which guide us in our management; they are:

(1) To prevent the vision in the deviating eye from deteriorating and to restore it when defective.

(2) To remove the cause by training the defective fusion faculty.

(3) To remove the disfigurement by bringing the visual axes of the eyes into their normal relationship.

The means we possess for carrying out these indications are six in number, viz.:

(i) Correction of the refractive error so as to make the vision in each eye equal.

(ii) Complete occlusion of the fixing eye so as to train the patient to use the deviating eye and restore its defective or lost function.

(iii) Instillation of atropine into the fixing eye only until both eyes are equally used in fixing.

(iv) Training of the fusion sense so as to teach the brain to blend into one stereoscopic picture the two images presented by each eye separately.

(v) Training the muscles of the eyes when any defect exists.

(vi) Operation to assist removal of the defective position when it cannot wholly be overcome by the above methods.

Except the first and last all the other means come within the power of the family physician to carry out, whether possessed of special ophthalmic knowledge or not. When the surgeon is fully equipped by knowledge, experience, and instruments the correction of the refraction will also come within his province.

The indications and methods being thus stated, it remains then only to learn how such are to be carried out.

Investigation of any case of strabismus should be made along the following lines :

(1) *History*.—Under this heading we should inquire regarding the age of onset, how it began, and any probable cause such as a preceding illness noted. Inquiry as to any history of squint in any member of the patient's family should not be omitted.

(2) *Nature of the squint*.—Whether convergent, divergent, alternating, etc., the cover and mirror tests already mentioned being employed in determining which of these conditions is present.

(3) *The state of central fixation*.—This point can be ascertained by the mirror as already mentioned. If, when the straight eye is covered, the unscreened one moves so as to bring the corneal reflex into the centre of the cornea, we learn that the power of central fixation is present, or, in other words, that the patient can still use the squinting eye for the purpose of seeing with. When the eye does not fix it has ceased to be used and is in a condition called "amblyopia ex-anopsia," or blindness from non-use.

(4) *The movements of each eye separately* should be tested by screening one eye whilst at the same time the patient is told to follow the movements of the mirror or finger with the uncovered eye. In very young children some bright object, as a bunch of keys, should be used instead, as of more interest than a finger or mirror.

(5) *The visual acuity* should be investigated. In older children the ordinary test type can be employed ; in infants the use of small ivory balls is perhaps the simplest means. One eye is covered up at a time and the child told to pick up the balls from the floor, and according to the way he sets about getting them judgment of the visual acuity can be fairly accurately gauged.

(6) *The refraction of each eye* is to be ascertained. For this purpose atropine, 1 per cent., is instilled, preferably in the form of an ointment, three times a day for a week. Glasses are ordered at the end of this time of a strength to within half a dioptre of the full amount. Whilst the patient is awaiting the glasses the mydriatic is instilled once a day only in both eyes so as to make it easier for the patient to become accustomed to the glasses gradually.

These outlines comprise the details of the first two visits. If desired the angle of the deviation can be measured by Worth's instrument, which is the simplest, or by Priestly Smith's method, or the perimeter.

All these details can be carried out if desired by an ophthalmic surgeon and the case then referred back to the sender for further treatment, which will consist in carrying out the following indications :

Occlusion of the fixing eye.—The idea aimed at here is to endeavour to make the patient use the squinting eye. In those cases in which central fixation is easily obtained it may only be necessary to instil atropine in the fixing eye, but in cases where it is very deficient it will be necessary to completely occlude the fixing eye. Frequently one sees patients going about with only a cap over the eye ; this is a perfectly useless proceeding, as such patients learn to look out beneath the shade in preference to using the squinting eye. To be at all effective the eye must be covered with a piece of lint which is held in position with strips of plaster. This occlusion is kept rigorously applied until we (by means of the mirror test) ascertain on taking off the cover that the originally squinting eye is used in preference to the other for *both near and far vision*. Such a result will naturally bring the deviation over into the other eye. The cover may require keeping on for a month or even longer at a time, it being taken off and the eye cleansed, and re-applied weekly. Of course the correcting glasses must be worn constantly, the occlusion pad being placed underneath the spectacles. When we have succeeded so far in transferring the squint we can leave both eyes open for a week and await results.

Instillation of atropine into the fixing eye only.—

This method is employed for the same purpose as the occlusive bandage. It is employed for those cases in which the power of central fixation is more equally distributed between the eyes. Thus it may in appropriate circumstances replace the occlusive pad, but is more generally required as a supplementive measure. We continue the instillation only just sufficiently long enough in the fixing eye to obtain fixation in the squinting eye. When this stage is reached, which is ascertained by the mirror test, the instillation is stopped for a week. Subsequent examination will reveal if either eye is preferred for fixation, and the instillation repeated in whichever eye is found to be the fixing one until a state of matters is reached revealing an ability to fix with both eyes at will whilst both remain uncovered. We shall have thus succeeded in producing an alternating strabismus in addition to having restored the vision in the originally squinting eye. We are now in a position to train the brain to fuse the two images.

Training the fusion faculty.—To carry out this part of the treatment we require to obtain Worth's amblyoscope, which is by no means an expensive instrument. In the employment of this instrument we must bear in mind that we have to develop and build up the function of a centre in the brain which is poorly founded. Were such not the case the occurrence of squint would not be possible. Further, the subjects we have to deal with are children, often of a highly sensitive and not too perceptive a nature. Their handling and successful management demand a combination of qualities in their teacher which, unless possessed of, spell failure from the start. Such cases are, therefore, at times trying, and call for great tact and perseverance combined with firmness. However, as a rule children soon learn to take a great interest in the exercises, and progress, once started, is both rapid and gratifying. It is due to the absence of some of these essentials that the procedure has in some hands not been successful. The method is correct—failure is due to the operator.

To carry out the amblyoscopic training the little patient wearing his glasses should be seated opposite the surgeon and in front of his eyes is held the instrument. On either side are placed two lights, which should be capable of being varied as regards their illuminosity. This can be accomplished in various ways, the simplest being to so arrange the lights that they can be brought nearer or moved further away from each end of the instrument.

Two of the pictures supplied with the instrument are then taken, one being inserted at either end. It is best to commence with those of the bird and the cage. When they are in position and the lights adjusted so as to illumine each, the little patient is asked to state what he sees. If the picture of the cage is opposite the originally fixing eye he will say that he sees the cage. The light which is illuminating this picture must then be reduced or even extinguished until the picture of the bird comes to be recognised by the other eye. Then by gradually increasing the illumination of the cage picture a stage will be reached when both pictures are recognised under equal illumination. All this means time and patience. The next step consists in approximation of the pictures by moving the tubes. At first, as the pictures tend to coalesce, the image of one picture will disappear. Practice and patience will in time overcome this until a stage is reached when the little patient exclaims that the bird has gone into the cage. At this stage we have obtained the simplest form of fusion or blending, and the condition can be strengthened by substitution of more and more complex pictures. So far we shall only have obtained fusion in the position of the eyes as they were. We must next endeavour to obtain a maintenance of the fusion throughout a fairly wide range of movement. To obtain this end we move by small steps the instrument, endeavouring the whole of the time not to lose the blended picture. With practice it will be found that this is soon accomplished, until a stage is reached when fusion of two dissimilar objects can be blended and maintained as one picture throughout a very large arc of movement. Once this is fully obtained we have overcome for once and all the chief cause of the squint, and in many cases these measures will have succeeded in bringing into correct relationship the originally defective position of the eyes. Once a week or once a fortnight for half-an-hour at a time is quite often enough for these exercises. It has been found by practice that nothing is gained by giving them oftener. One must not omit to mention that there are certain circumstances which preclude all possibility of ever obtaining a true fusion faculty. Such occurs in the type of true alternating strabismus and in cases in which treatment has not been commenced before six years of age.

Up to this stage we have accomplished the following: We have made the vision equal in both eyes by glasses, restored the function of observation to the squinting eye by the occlusive bandage, and with the assistance of atropine adjusted the work of the eyes so that one is not employed more than its fellow. Lastly, by means of the amblyoscope we have developed the receptive centre in the brain, at first by obtaining simultaneous recognition of two dissimilar images approximating same by mechanical means until a super-imposition leads to a true blending or fusion. Such fusion once obtained is cultivated until it can be maintained whilst attempts at separation are made by the instrument originally used for blending.

Such, then, is briefly Worth's principle of treating strabismus, and in the majority of cases of the convergent type, which is the commonest, no further interference is required. It is, moreover, sound in principle, scientific in theory and practice, and marks one of the greatest advances made in ophthalmology for many years.

There are, however, some cases which cannot by these methods alone have the unsightly appearance of the squint entirely removed, and further improvement can be obtained by Maddox's instrument for strengthening the weak muscles or by operation.

Maddox's instrument, which he calls a prism verger, is based on the principle of making the patient maintain binocular fusion whilst prisms are rotated in such directions as will necessitate the bringing into action the defective muscles. Such exercises demand naturally that the fusion faculty should be completely developed, otherwise double vision would only too readily be obtained and our efforts nullified.

To use the instrument the patient should be seated at 5 or 6 metres away from a set of test type. He should be told to maintain his attention on the smallest of the letters which he can read distinctly through his spectacles. The apices of the prisms are set in the instrument in the position which gives him binocular single vision: for an internal squint the apices of both prisms would be set inwards, and for a divergent strabismus outwards. By rotating a milled head the prisms are moved so as to cause the apices to rotate in the direction in which the movement of the eyes is defective. This for an internal squint would be outward, and in a reverse direction for a divergent strabismus. The rotation of the prisms is proceeded with slowly and gradually until a stage is reached when the patient declares that the letters are growing indistinct. A pause is then made whilst the patient by an effort of will obtains clearness of vision. Rotation is resumed and another rest given until a point is reached at which no further clearing can be obtained and the patient confesses to the presence of a doubling of the letters. Repeated exercises varying in time and frequency according to the requirements and results obtained in each case soon give evidence of brilliant results. In young patients at the beginning of the course of exercises no blurring of the letters may be complained of during a very extensive rotation of the prisms. This is due to the patient preferring the image of one eye to the other and not observing the doubling of the letters. The presence of such is easily discovered by resorting to the cover test. If either eye be covered by a card or screen when only one eye has been in use, a movement will take place in the uncovered eye. Had true binocular single vision been present it is obvious no movement would be necessary on the part of this eye. Where this condition of affairs is present resort must be had once more to the amblyoscope so as to train the maintenance of the fusion faculty. Finally comes the question of operation. This ought to be the very last resort, and not, as has been in the past, the first and most abused remedy. Here, again, just as much scientific improvement has taken place in the technique of the operation as has attended the pre-operative training methods. Haphazard severing of muscles is, or ought to be, as much a thing of the past as indiscriminate venesection. Before operating careful examination of the degree of the deviation should be made and steps adopted to ascertain how much is due to muscular inefficiency and how much to hypertrophy of the opposing muscle. For the former endeavours would be made to develop the weak muscle by appropriate prism exercises, and later its advancement with or without shortening could be done; for any hypertrophy we would have to choose between a partial central tenotomy or lengthening of the muscle according to one of the various methods now so well known. Under certain circumstances a combined advancement and tenotomy would be adopted. Simple tenotomy as a rule is seldom required, for not only does it entail a serious risk of falling back of the muscle with the more than possible chance of leading to an opposite condition of the squint, but precludes any use being made of the cut muscle for operation at some other time. Again, tenotomy, by its effect on Tenon's capsule, causes a protrusion to take place forwards of the eyeball, which results very often in a most unpleasant appearance being given to the eye, and finally not only is the operation unscientific owing to the uncertainty of its ultimate results, but it weakens a function of the visual apparatus which may lead to serious results in later life.

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MEDICAL CASES IN SURGICAL PRACTICE.

A Clinical Lecture delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

I ASSUME that throughout your careers you will be brought into contact with a great variety of diseases, and that you will have to determine whether they belong to the category of those called medical or to those we call surgical, and which may, therefore, require to have a surgical operation performed, or, at all events, have some form of surgical treatment. A case which is sent to you already "labelled" is sure to be full of pitfalls. I suppose you, like myself, cannot help thinking that when a patient is sent with a sarcoma of the chest wall that the probabilities are that he has one. Further, if he seems to have a sarcoma of the chest wall you will get an obsession that it is a sarcoma of the chest wall, and possibly perform an operation for sarcoma of the chest wall, although, after all, the mass may turn out to be inflammatory, with a chronic abscess behind it. Now such a case is difficult, but gives you time for examination, and for repeated examinations. It gives you time for much inquiry and it gives you time for much thought. But often cases will come before you which have acute manifestations and which seem to want immediate surgical treatment and which may get immediate surgical treatment, consisting of opening and exploring the abdomen, when, after all, the disease was one which was not likely to have been benefited by the operation, not likely to have been even diagnosed by the operation, and which was very likely indeed to have been made very much worse by the operation. Now I am sure you will agree with me that it is a lamentable thing to perform an unnecessary operation. It is foolish, and if it is done without sufficient

care and attention it is almost culpable. I am about to try to warn you that these things occur. A philosophical historian has said that we should learn first of all to doubt, because doubt leads to inquiry, and inquiry leads to discovery. And if you feel doubt about every case which is presented to you I am sure you will not fall into some of the pitfalls that others have fallen into.

It is necessary to arrange my material in some kind of order, and I will first of all mention cases in which the manifestation was in the abdomen whilst the disease was in the nervous system. A most striking instance of this was under my care about three years ago. A man was admitted because he was suffering from repeated attacks of vomiting. Those attacks of vomiting had continued from the year 1900. When first they began they were accompanied by pain in the right iliac fossa. What other manifestations he had at that time I cannot tell you; I did not see him. But the vomiting and pain in the right iliac fossa led a surgeon to remove his vermiform appendix. He recovered from the operation, but derived no benefit of any kind whatever. He was admitted into this hospital four years later and had frequent vomiting. At that time he had persistent pain in the abdomen between the umbilicus and the sternum and the pain was said to pass down the left side of his abdomen. It stated in his notes that he vomited most of the day, so clearly the vomiting was an urgent sign. At first we surmised that his vomiting might be due to disease of the stomach, and therefore he was examined with the view of seeing whether he had any growth at the pylorus, or whether he had any other disease which would make it worth while for him to undergo an operation, such as gastro-enterostomy. His stomach was but slightly dilated, for when distended with air it merely reached the umbilicus. In addition it was found that its peptonising powers were not impaired, and that that which was extracted from it was normal. After he had been in the hospital for a while the vomiting ceased, but the pain at the pit of the stomach persisted. And now another question arose about the diagnosis and another question as to surgical treatment. He was now examined to see whether these symptoms were due to chronic inflammation of the pancreas. At about that time, March, 1904, a great deal was written in the press concerning chronic pancreatitis. New and elaborate methods of examining the fæces were being devised with the view of placing the diagnosis on a scientific basis. There were some points which might have led one to suppose that the pancreas was not functioning properly, because when he had oily material given him, and particularly when he had castor oil, it passed through unaltered. The Pathological Department of the hospital kindly undertook to make that very elaborate test, which was devised by an old Bartholomew's man, Dr. Cammidge, and they reported that the result of their examination indicated that there was impaired function of the pancreas due to past disease; but they said that their examination helped to exclude the possibility of malignant disease, and likewise the possibility of acute pancreatitis. They sent up a very full and very elaborate report. After this the vomiting began again, but I could not see my way to doing this man a good turn by draining his pancreas. You are aware, of course, that it is not unusual, when a patient has an acute or even chronic inflammation of the pancreas, to endeavour to drain the pancreatic ducts by placing a drainage-tube into the gall-bladder. I did not think it worth while to do this for our patient. But I did think it worth while to have the man kept under observation after his return from Swanley. And I remember very clearly someone coming to me with great triumph to tell me that the man had returned with the most unmistakable signs of locomotor ataxy. He was seen afterwards, and there was no doubt that he had locomotor ataxy; and there was no doubt left in my mind that all his symptoms were referable to that disease of his nervous system, and that they were not due to disease of his stomach or pancreas. That ataxic person had had his appendix removed—evidently an unnecessary operation—and he stood a very good chance of having gastro-enterostomy performed, and he also stood a very good chance of having his gall-bladder drained. I cannot explain to you at all why one felt so suspicious about that man. It is difficult always to give reasons for the belief which is in one, but I suppose that at the time one had reasons in one's mind, probably founded upon examination of the whole patient.

There has been recently under my observation another man whose history is rather curious. This person had had a Symes' amputation performed.

I do not know why a Symes' amputation was performed. I believe it was done for tubercle of his foot. After that he had persistent pain in the posterior tibial nerve. I remember exposing his posterior tibial nerve above the stump, and cutting about two inches out of it to see whether I could stop the pain. However, it is clear that it did not stop the pain in his nerve, and I afterwards, with the assent of my colleagues, performed amputation of the leg higher up, at the junction of the upper and middle third. That relieved his pain, and he disappeared from my ken for some years—three or four years probably. But he has been in the hospital within the last two months, because the pain had returned in his stump. But now an alteration had taken place, because it was clear, from the examination of his eyes and the condition of his equilibrium, that he had locomotor ataxy. He was then treated by intra-muscular injections of mercury—a treatment which I earnestly commend to your notice. When last we saw him it was obvious that he had undergone very marked improvement. Operations for the relief of pain are rather speculative. I wonder if you remember me telling you about a patient who had had her rectum removed for malignant disease, but who afterwards became afflicted with intolerable pain down the left leg, like the pain of neuritis. I made an incision over the great sciatic notch, and passed my finger through and felt malignant disease inside growing into the sciatic nerve. I freed the sciatic notch, and for a time that patient was relieved. I tried to cure that patient of her pain by exposing the posterior roots of her lumbar nerves and removing them. That cured her of the greater part of her pain. But I then discovered that it had not cured the pain in the joints. And I was then informed that the sensation of the joints passed up the anterior roots of the nerves. Another patient who had had her breast removed had horrible pain, and I performed inter-scapulo-thoracic amputation of the shoulder. And I observed then that the malignant disease had grown along the course of the brachial plexus. I removed the brachial plexus as close to the vertebral column as I could, but she continued to have severe pain in the first dorsal nerve. Now more than once I have been requested to see patients with the view of saying whether the pain caused by inflammation of the nerves of the limbs was due to malignant disease. I do not know whether any question of surgical interference arose, but clearly the question of surgical interference might arise.

Now I will pass on to another kind of medical case which may be operated upon unless you are upon your guard. I remember very well receiving a letter in the middle of the night requesting me to see a man with acute intestinal obstruction. He had absolute constipation, was passing neither wind nor fæces, and was vomiting. I did not go down because I saw at the end of the letter a note which said he was recovering from gonorrhœa. I knew that people who were taking oils, such as copaiba, for gonorrhœa, often suffer from the most violent pain in the abdomen, that they cease to pass any wind or fæces, and may vomit. Therefore I guessed that this man was suffering from something which he had taken for the cure of his gonorrhœa. I saw him the next morning. He was then better and produced from his locker a bottle which I suppose contained copaiba. I remember one other case of a similar character. I performed the radical cure upon an officer, and after he had left my care he went to stay at an hotel. I received a very urgent message to go and see him one day because he said that whilst he was straining at stool something had gone wrong in his inside, that he had suddenly been seized with the most violent pain in the abdomen, and had got obstruction and some distension. When seen it was clear that he had constipation and had not passed wind for some time. He had some distension and also was suffering from great pain in his abdomen. At first glance it seemed that something might have gone wrong in the region of his operation. However I had heard him casually remark to a friend that he could not partake of any alcoholic beverages because he was suffering from inflammation of the kidneys. I thought that was curious, because I knew he had not inflammation of his kidneys. I said to him: "What was the meaning of the inflammation of the kidneys which you talked about the other day?" He said: "I have just recovered from gonorrhœa." "What are you taking for it?" And he produced capsules which he said contained copaiba. I concluded that the bout of obstruction, distension, and pain were due to the copaiba which he was taking. In future when you know that a patient with such symptoms is taking these oils you will not fall into the trap

and send for a surgeon to operate for acute intestinal obstruction. I can recall the case of another patient which puzzled me very much at the time, and which had very curious features about it.. I was sent for rather late one night to see a young man who was supposed to be suffering from acute appendicitis. He had seen a physician of repute, who said that he thought a surgeon should see him with the view of that question being taken into consideration. This patient had acute pain and great tenderness in the right iliac fossa. He had a temperature of 103° F. and a pulse of at least 120. He seemed exceedingly ill, and I can remember now the curious sensation which one felt on passing the finger into the rectum to ascertain the condition of his pelvic peritoneum, as of the presence in Douglas's pouch of a contracted and empty small intestine. I felt very much inclined to think that he was suffering from an acute attack of appendicitis, but although he had pain and tenderness in the right iliac fossa he had a good deal of tenderness in other parts of the abdomen; and if he had a temperature of 103° F. and a pulse of 120 caused by an appendicitis, that appendicitis must have been intensely acute. And I thought the area and degree of tenderness did not quite coincide with the severity of his general symptoms. So I advised that the patient should be placed under proper observation, and did not then think it necessary to perform an operation. I saw the patient again, and by that time it had become clear that he was suffering from an exceedingly acute attack of typhoid fever, and after a while I regret to say he had more than one violent hæmorrhage and died. Yet that was a case in which a physician of great experience and repute had taken into consideration the possibility of that man having, not typhoid fever, but an acute attack of appendicitis. Such cases will occur to you, and if there is not an epidemic of typhoid in your district at the time and if the possibility of typhoid is not clear to your minds, it will be easy for you to fall into a similar trap.

We have recently had in our wards a very good example of another kind of deception. A little girl was admitted a few months ago suffering from exceedingly acute abdominal pain. That pain was felt in, and referred more particularly, to the right side of the abdomen low down. At the same time she was vomiting and lay on the bed with her legs drawn up. When I saw her the temperature was 101° F., the pulse 136, and the respirations 34. She was very constipated. But, looking at the whole patient, I did not think that the pain in the right side of the abdomen, the tenderness and rigidity there, and the vomiting and constipation could be due to an attack of appendicitis. All the points are not clear in my mind which led me to the conclusion that at all events she did not require immediate operation for acute appendicitis. And I also knew this: that that child was in St. Bartholomew's Hospital, that she was under constant observation, and that if any change took place in her condition, if she got any more of those determining symptoms of which I have spoken to you before, there would be no difficulty in her receiving immediately the surgical assistance which she might require. However, the vomiting ceased, the bowels acted after an enema, the abdominal pain and rigidity diminished, and after forty-eight hours she had obvious signs of pleuro-pneumonia on the right side, and so she escaped the needless operation for appendicitis. I believe that not a few have been performed for this condition. In pleuro-pneumonia I have seen pain and rigidity of the abdomen on the left side. I operated upon a young lady and removed a chronically inflamed appendix with concretions in it, so there was no question about the diagnosis. But about a week after the operation she became very ill with an attack of influenza. In a little while she began to vomit, and at the same time had intense pain and rigidity of the abdominal wall, with tenderness over the region of the stomach. So it was supposed she had a gastric complication or that she might have got peritonitis. However, I suspected, as she had had influenza, that she had got pleuro-pneumonia, and in a little while she began to spit up the characteristic sputum; and my colleague, Dr. Norman Moore, saw her and confirmed the diagnosis. She made an uneventful recovery.

I might narrate to you several other cases of pleuro-pneumonia accompanied by abdominal manifestations, but if you are constantly on the look-out for the abdominal manifestations of pleuro-pneumonia I do not think anybody here will be deceived by them.

I have not time to mention the numerous instances in which mucous colic or muco-membranous colitis have been mistaken for appendicitis and operated upon.

I will now mention to you another class of medical case which is sometimes operated upon. I remember assisting at an operation upon a man who had had violent pain in the left kidney, tenderness in the left flank, and considerable hæmaturia. It was concluded that all these symptoms were due to the presence of a stone in the left kidney. Accordingly the left kidney was exposed and explored. It was intensely inflamed and bled a great deal, but contained no trace of stone. The hæmorrhage at the time of the operation was considerable and continued after the patient went back to bed. Therefore the surgeon and myself went upstairs to see the patient, and I remember the first question the man asked was—he was suffering from the effects of shock and hæmorrhage, but was quite rational—"Where is the stone?" I mention this because if you speak of a stone the patient will expect to see the stone. I am usually cautious about what I say to patients who are supposed to have stone. I more usually say: "You have some condition of the kidney which makes it worth your while to have an operation performed upon it. If there is a stone it will be extracted, but you may have some other condition which can be put right." And I have often been thankful to have given my advice in such a guarded manner. But this man succumbed, and it was afterwards found that he was suffering from acute nephritis. I do not know how that calamity could have been averted. I may be wrong, but I cannot help thinking that a very persistent and rigorous microscopical examination of the urine may have been omitted. I think that such acute nephritis as that man had was almost certain to have led to casts appearing in the urine, and so an operation might have been avoided. But there has been a still more extraordinary occurrence in connection with nephritis, and I am permitted to mention the following case. A patient was admitted not very long ago who had marked symptoms of intestinal obstruction. He passed neither wind nor fæces, he was vomiting, the abdomen was distended, and clearly he was critically ill from some form of intestinal obstruction. The obstruction was complete. The usual test was applied. If there is any question in your mind whether a patient has intestinal obstruction or not, I advise you to give what I call test-enemata. And be very careful when you give these test-enemata. I do not think it matters what they are made of providing they are made of something irritating, that they are copious, and that they are given in the presence of the medical man himself. Otherwise air may be injected with that rather dangerous instrument, a Higginson's syringe, and lead you to suppose that the obstruction is not complete, and so lead to delay. Four test enemata were given to this man and nothing ensued, and, very properly, I think, his abdomen was explored to try and ascertain the cause of the obstruction. A loose body was found in the right iliac fossa, and that is rather curious, but in the post-mortem room and in the dissecting room such loose bodies are sometimes found. The cause of the obstruction was not discovered. The man returned to bed and recovered in a most satisfactory manner from the abdominal exploration. But in twenty-four hours he began to pass urine, which was acid, had a specific gravity of 1016, and contained albumin and casts. So there was no question then but that he was suffering from some form of nephritis. I do not know how a person with acute nephritis can get an attack of intestinal obstruction, but this man certainly did, and I see here that the diagnosis of nephritis was confirmed by a post-mortem examination. I have recently seen another case of a similar kind, but no operation was performed.

I have often wondered what is the meaning of those cases which one occasionally sees reported. Am I going beyond the strict truth when I say that I have seen a heading of this kind in one of the medical papers: "Intra-capsular Fracture of the Neck of the Femur; Acute Intestinal Obstruction; Laparotomy; Death." I think to have seen such a heading, but still you must know that when an old lady fractures the neck of the femur and is on her back you should look forward to, and forestall, a bad attack of flatulence. And I have often wondered whether the gentlemen who record these curious cases have not mistaken an exceedingly acute attack of flatulent distension, which might have been relieved by enemata, for intestinal obstruction. I think if you are warned against such an event you will not fall into error.

I have just time before ceasing to call your attention to another class of medical case which to me seemed as full of deception as any case possibly could be. I recall very clearly being asked to see

a gentleman whose age was about seventy, because he had acute suppurative arthritis of his knee, and it was thought that he might require to have amputation performed. I saw a most intense inflammation, swelling, and œdema in and outside the left knee-joint. The skin over the swelling was vivid red, intensely painful, and very tender. The muscles of the thigh and leg were wasted, the temperature was high, the pulse accelerated. The slightest movement caused anguish, and I felt very much inclined to agree with my friends that the patient was suffering from an acute septic arthritis in the left knee, and I certainly thought that amputation might have to be done. But there were one or two curious features about the case. If a person has acute septic arthritis of the knee, when you come to look at the whole patient you see that he looks very ill; in addition he will probably have had a rigor, and be listless, weary, and not desire to move; he perhaps cannot take anything to eat or drink, and is very ill. Now, this struck me about that gentleman: that if he had acute septic arthritis of his left knee instead of being ill and depressed he was in an appallingly bad temper, and extraordinarily vigorous. So his knee was placed upon a suitable splint, was watched for a little time, and in the end we agreed that he was suffering from an exceedingly acute attack of gouty arthritis, and for that he was treated. And after a long illness, and after a long period of massage for the wasted muscles, he got quite well and was able to walk about, and returned to his usual health. I may be wanting in intelligence, but am quite prepared to admit that when first I saw that knee I thought it was a case which would require amputation of the thigh. I have seen one other case of acute gouty arthritis in which I was called in to see the patient because it was said by the physician in charge that it was either a case of gout or of acute septic arthritis. There was an œdematous foot, an ankle swollen with intense inflammation inside and outside the joint, and there was redness and œdema passing up the inside of the leg, but there again the general symptoms did not coincide with the local symptoms. And, perhaps taught by experience, I was able to tell the patient, who was a friend of mine, that he had the second worst attack of poor man's gout that I had ever seen. He, too, escaped the suppuration which was supposed to be impending, and after a rather long illness got perfectly well, and now has a good, sound, healthy ankle-joint.

I will cease with the narration of these cases. I hardly think it can be necessary to tell you that I have occasionally seen acute suppurative periostitis called rheumatism, and treated with salicylates until there has been a considerable stripping off of the periosteum, with subsequent necrosis. Such a difficulty I do not think ought to happen to anybody who has been educated in this hospital. However, you must be on your guard and remember that such cases will come within your cognisance.

I could, of course, deliver many lectures on a topic such as this, and warn you against the dangers of confusing other medical diseases with surgical conditions. But you will pass all the rest of your lives in learning not to fall into these pit-falls of which I have warned you. I wish to thank Mr. Trapnell for having given me notes of a number of the cases which have been under our care.

November 2nd, 1908.

Nephritis of Pregnancy.—Baumann considers that, in the kidney of pregnancy and the nephritis of pregnancy, so long as there is no eclampsia, the prognosis for the mother is comparatively favourable. He has records of only two cases in which the renal affection became chronic. The maternal mortality is slight. No patient was operated on on account of threatening symptoms. The fœtal prognosis is more unfavourable, because frequently premature labour results and the child is either born dead or without vitality on account of the lack of development. In his cases the fœtal mortality, when no eclampsia occurred, was eight out of forty-seven, in other words, 17 per cent. The therapy of nephritis of pregnancy and pregnancy kidney consists in rest in bed and a milk diet, as long as no other threatening symptoms are manifest. If the patient is drowsy, restless, etc., then hot baths should be ordered, likewise hot packs, in order to promote diaphoresis. Lastly, in threatened eclampsia, chloral hydrate and chloroform are indicated. Chloral hydrate in a dose of one or two grammes *per rectum* and inhalations of chloroform, accompanied by the usual therapeutics of nephritis, represent all that can be done.—*Journ. A. M. A.*, vol. li, No. 15.

THE VALUE OF AN EXCLUSIVE PROTEID DIET IN CERTAIN DIGESTIVE DISORDERS.

By ERNEST YOUNG, M.D.

ALTHOUGH attention has frequently been directed to the therapeutic value of an exclusive proteid diet, to wit, a minced-meat and hot-water diet, in obesity and certain conditions of chronic disease, its efficacy in digestive disorders has not received that recognition from the profession at large which it undoubtedly deserves.

Saundby (1) pertinently supports this view when he says: "The digestibility of finely-divided meat is not sufficiently appreciated; probably no food is tolerated so well by our stomachs, for even where the gastric chemistry is deficient such food is easily got rid of and causes no discomfort."

This lack of appreciation, however, is not confined to those who do not prescribe the diet, since many who may have employed it successfully in cases of gout, chronic rheumatism, etc.—where it will often succeed despite faulty technique and administration—may, nevertheless, have met with only partial success, or even apparent failure, when applying it to digestive disorders pure and simple, owing to the fact that in these conditions details of preparation, administration, and careful regulation of the diet to the digestive capacity of the individual (determined by clinical methods which will be described later) become of paramount importance if a successful issue is to be looked for.

This neglect of detail arises from the limited conceptions which prevail concerning the diet, a not infrequent idea being that the sum total of its rationale consists in the giving, to each patient alike, of half a pound or so of minced beef three times a day and a pint of hot water four times a day. The folly of prescribing a diet in this routine fashion without regard to the individual, particularly when the individual is the subject of digestive disorder, should be obvious, but, even taking for granted that the diet has been appropriately regulated to meet the needs of the particular case, the good results which may be anticipated are frequently stultified by inattention to the other details mentioned, such, for instance, as the proper preparation of the meat (for which the physician should be able not only to give adequate instructions, but also be able to judge by inspection and taste whether these instructions have been carried out efficiently), the correct temperature of the hot water, and the hours at which the meat and hot water respectively should be taken. This last detail is a frequent source of error, the water being given shortly before, after, or even with meals, instead of at least one clear hour previously, so that it may have time to cleanse and be evacuated from the stomach before the subsequent meal.

It must be admitted that this neglect of essentials is largely due to the scarcity of definite literature upon the subject, even Dr. Salisbury, the originator of the system, paying little or no attention in his text-book (1) to digestive disorders pure and simple. I purpose, therefore, in the present article to endeavour to remedy this deficiency by briefly describing the rationale of the diet, or, rather that adaptation of it which I have found to be the most suitable in dealing with these conditions.

Before doing so, however, I would emphasise the fact that the diet must be regarded as a therapeutic measure only, that is, simply as an *abnormal* diet, often peculiarly suitable in helping to restore an *abnormal* condition of digestion to the *normal*. Once this result is attained the patient should, with rare exceptions, be transferred to an ordinary mixed diet of proteids, fats, and carbohydrates with as little delay as possible.

Cases Suitable for Treatment.

It need hardly be mentioned that such a rigid *régime* is seldom, if ever, required in mild cases of functional dyspepsia. If adopted in cases of moderate severity, a modified form of the diet will usually suffice to remedy matters.

Speaking generally, however, the diet should be reserved for chronic digestive disorders such as chronic gastritis, chronic hyperchlorhydria, dilatation of the stomach, etc., which persistently resist the usual lines of treatment. In the large majority of these cases the rigid diet should be administered *ab initio* if satisfactory results are to be obtained.

The Exclusive Proteid Diet.

The basis of the exclusive proteid diet consists of thoroughly cooked minced lean beef (so

prepared that all gristle and the greater part of the connective tissue are removed) and hot water. (Minced mutton or chicken may be substituted occasionally for variety, but bulk for bulk neither is so nourishing as the beef.)

The minced beef is given three times a day, with a five-hourly interval between each meal. The best hours for meals are 8.30 a.m., 1.30 p.m., and 6.30 p.m. In any case the last meal should not be later than 7 p.m.

The hot water is given four times a day, one and a half to one and a quarter hours before each meal, and three and a half hours after the last meal. The water should be *sipped slowly* at a temperature of about 120° F.—roughly that at which tea or coffee is usually taken. The quality of the water is an important factor, and if at all hard distilled water must be used.

The quantity of meat at each meal varies from two ounces upwards, the quantity of each portion of hot water being a pint, or less, according to the nature of the individual case.

Such is the exclusive proteid diet, *which is continued as long, and only as long, as the necessity lasts.*

Its therapeutic action may now be briefly considered.

Therapeutic Action of the Diet.

(A) *The hot water.*— The hot water, slowly sipped, performs many useful functions, in addition to acting as a general stimulant. Thus, taken at the times indicated,—

(1) It washes the stomach and stimulates it to expel gas, mucus, and stagnant contents, thus leaving it clean and in' good condition for the subsequent meal, the last quantity of hot water performing the same function previous to sleep;

(2) It acts as a stimulant to the skin, liver, and kidneys, promoting perspiration, increasing the flow and fluidity of the bile, and the quantity and solvent power of the urine;

(3) It increases the fluidity of the blood, thus tending to dissolve abnormal products which may have accumulated in the capillaries and lymphatics;

(4) Lastly, it acts as an efficient protection to the liver and kidneys by keeping in solution, and so rendering easy of elimination, any imperfectly oxidised products of nitrogenous waste which may result from the increased proteid ingestion.

(B) *The minced meat.*—With regard to the minced meat, it is obvious that—

(1) It contains a maximum of nourishment with a minimum of weight and bulk.

(2) The smallness of weight and bulk imposes but little strain on the motor power of the stomach, while the comminuted condition of the meat renders it easily accessible to the gastric juice.

(3) Being largely digested in the stomach, it gives temporary rest to the succeeding portions of the alimentary tract.

(4) Indirectly it tends to arrest the formation of toxins and consequent auto-toxæmia, proteid putrefaction being discouraged by careful quantitative regulation of the minced meat, while acid fermentation is inhibited by the absence of carbohydrate food.

As already mentioned, the rigid diet described is continued as long, and only as long, as the necessity lasts, this necessity naturally varying in accordance with the particular case. As the patient improves, a little supercooked starch food may be added, such as baked bread (thin slices of bread baked in the oven until golden brown), breakfast biscuits, rusks or zweiback, with which a little butter is sometimes allowable. Small portions of fish, chicken, fresh game, lamb, and mutton (not necessarily minced) may also be gradually introduced. Later, boiled rice, milk puddings, purées of vegetables and fruits may be added to the dietary (all such conditions, however, being justified by periodical clinical analyses), and so the patient gradually returns to a normal mixed diet.

Clinical Measures for Regulating the Diet.

The diet, as I have indicated already, should not be given in a routine manner, but regulated in accordance with the digestive capacity of the patient. This may be determined by analysis of the excreta, and must be regarded *as of the utmost importance* and value if the patient is to obtain the full advantage from the diet. Salisbury invariably examined the urine and fæces of his patients, both previous to, and periodically during, treatment. While by no means agreeing with all of Salisbury's opinions, I can emphatically endorse the practical value of this measure, for by thus obtaining definite clinical data regarding the digestive capacity and general condition of the alimentary tract, the physician is enabled to prescribe a suitable initial

diet, determine when other food may be added with advantage, and apply medicinal treatment in a rational manner.

Of the clinical methods mentioned simple urinalysis is universally understood, but the same unfortunately, cannot be said of the fæces, and it is to be regretted that Salisbury gave no definite instructions in his text-book as to the technique he employed with regard to it. I have shown elsewhere, however (3), that fæcal examination of a comparatively simple nature—such as may be undertaken within five to ten minutes by any medical man with an elementary knowledge of the microscope—is all that is necessary to provide adequate data on which to base suitable dietetic treatment.

To that article the reader is referred for complete and illustrated details of the method, space not permitting their reproduction here. In this paper only a brief general idea of the value of the analyses of the excreta in regulating diet can be given, this object probably being best attained by quoting a practical example, bearing in mind, however, that the analyses in the case to be described must only be regarded as fairly typical, inasmuch as analyses, even from apparently similar cases, are seldom quite alike in character.

The patient is a man, æt. 35 years, has suffered from chronic gastritis for many years, paid no particular attention to his diet, has gradually declined in weight and strength. Physical examination reveals tenderness in epigastrium, no apparent atony of stomach, no cardiac trouble, and (as proved by subsequent urinalysis) no renal disease. The urine is deep yellow in colour, specific gravity 1032, urea increased to 4 per cent., no albumen or sugar.

The fæces are markedly acid in reaction, and, microscopically, show defective digestion of all kinds of food, viz. undigested clumps of muscle-fibre, some undigested starch nodules, large patches of the gluten layer of wheat with gluten granules *in situ*, droplets and bile-stained masses of fat. Liquefied mucus, swarming with bacteria and gas-bubbles, is also present.

From the above analyses it is evident that, whatever the condition of the stomach may be, the intestinal digestion is seriously at fault, the process of abnormal fermentation and consequent formation of large quantities of organic acids rendering useless for nutrition a considerable portion of the food ingested, in addition to irritating and impairing the selective power of the intestinal epithelium and so allowing toxic material to be absorbed.

The advantage of placing such a patient upon an exclusive proteid diet of bland nature and hot water must be obvious. Thus, the absence of the offending carbohydrate foods will, by inhibiting abnormal acid fermentation, tend to eliminate irritation, catarrh, and auto-intoxication; the exclusive diet of minced beef will give but a minimum of work to the stomach and intestines, and so promote much-needed rest; while the hot water will tend to cleanse away existing catarrh and help to dissolve, and so render easy of elimination any toxic material in the blood-stream.

Such a patient, then, may be placed upon three or four ounces of minced beef at each meal and a pint of hot water four times a day, analyses of the excreta being performed again in a week's time. If matters have gone well the urine will be paler in colour, less acid in reaction, specific gravity lower, and urea output nearer the normal. The fæces will probably be black and sticky (due partly to the exclusive meat diet, partly to increased flow of bile) and will be less acid in reaction. Microscopically, they may still show traces of acid fermentation, but the meat should be almost perfectly digested or show only a few isolated muscle-fibres.

If, however, the initial quantity of meat was larger than that suggested, and has, therefore, not been adequately dealt with, the fæces will be alkaline in reaction (owing to proteid putrefaction) and will show undigested clumps of muscle-fibre, while the urine, in addition to having its chief abnormal features unchanged, or perhaps accentuated, will show marked indicanuria.

We will presume, however, that the quantity of meat was carefully regulated and that matters have gone well. This being the case, half to one ounce of meat may be added at each meal. Provided also that the periodical analyses are satisfactory, a similar addition may be made each week until six to eight or ten ounces are reached; more than this is seldom required. If the patient tires of the minced beef and wishes for something to chew, a few ounces of fish, chicken, lean mutton, etc., may be taken as part of a meal *once* a day.

The question now arises, How soon may starch or other food be introduced with advantage? and

may be answered as follows: When from a week to a fortnight the urine has been normal in colour, output of urea, and with a specific gravity of 1020 or less, and when, in addition, the reaction of the fæces is normal (that is, faintly acid, faintly alkaline, or amphoteric), and they show, microscopically, a perfect digestion of the meat food and no traces of abnormal fermentation, then, and not until then, should starch food be introduced. This, in a case of long standing, may be anything up to six weeks, or even more, from the commencement of treatment. In a case of moderate severity, however, a little starch food may usually be allowed in from two to three weeks. Begin with half an ounce to an ounce of supercooked starch with each meal. If this agrees, a little fresh butter may be allowed with it the following week. Provided all goes well for another fortnight or so, a table-spoonful of boiled rice or macaroni or milk pudding may be added at two meals a day. During this time the meat portion of one meal each day may be partly supplied, as already indicated, by fish, chicken, mutton, etc. Green vegetables and fruits may be introduced later, but generally it is not advisable to give them until the patient has progressed favourably on meat and starch foods for some weeks, and then give them at first only in the form of purées. The above order of increasing a patient's dietary is the one I have found to be the most suitable for the majority of cases. Occasionally, however, patients are able to take green vegetables and fruits with impunity before starch foods can be tolerated; others, again, have always an idiosyncrasy for butter. No hard and fast rule, therefore, can be laid down; the one reliable test must be periodical analyses of the excreta and *diminution*, *deletion*, or *increase* of food according to the results.

I may be told that numerous cases have been cured by the diet irrespective of these analyses. This I admit, but maintain, nevertheless, that but for the neglect of analyses of the excreta the majority of these cases could have been cured more speedily, while many of those cases in which the diet has *apparently* failed would have had a successful issue.

I cannot refrain from suggesting here that some system of fæcal examination should form part of the armamentarium of every physician who is called upon to treat digestive disorders, whatever may be his opinions concerning diet. This applies particularly to those cases where intestinal as well as gastric derangement exists, as it so frequently does, in chronic digestive disorders, in which analyses of the gastric contents can only be of partial value. It is no exaggeration to say, however, that in the large majority of cases, neither gastric nor intestinal analyses are performed, patients being treated merely from subjective symptoms, combined, perhaps, with an external examination of the stomach. Subjective symptoms must necessarily be of very limited value, depending largely as they do upon the degree of neurosis present; but, even apart from this, it is of frequent occurrence to find from clinical analysis that foods which patients stated suited them the best are, in reality, the chief cause of their trouble.

Conditions necessitating Modification of the Diet.

The details I have given regarding regulation of the diet are applicable generally to nearly all types of chronic digestive disorders, but a few conditions which necessitate some modification of the diet must be briefly considered.

(A) *Marked atony and dilatation of the stomach.*—If either of these conditions are present the hot water must be curtailed, since the bulk of a pint would obviously be injurious to the weakened viscus. In atony it is usually sufficient to reduce the quantity to three-quarters or half a pint, but in marked dilatation even this is frequently too much. If such is the case give half a pint in two portions of five ounces each, the first portion two hours, and the second one hour, before meals. The urine should be carefully watched, and if the above quantities do not suffice for the physiological needs of the patient they must be supplemented by enemata. If these modifications are carefully observed there is no disease in which the diet will give more brilliant results than dilatation of the stomach, and it is advocated for this condition by many physicians who do not prescribe the diet more generally. I am referring now to atonic dilatation; but even in obstructive dilatation—whether benign or malignant—where operation is refused or is not feasible, a diet consisting chiefly of the minced meat will prove most helpful, owing to the ease with which it is evacuated from the stomach.

(B) *Chronic hyperchlorhydria.*—This condition, admittedly one of the most difficult to cure by any

form of treatment, occasionally requires careful adjustment of the diet. As will, perhaps, be anticipated, these cases are able to take relatively large amounts of meat with impunity, and, provided no marked motor deficiency exists, they may be started on eight ounces at each meal, and this quantity may usually be increased fairly rapidly up to ten or twelve ounces. Not infrequently immediate improvement is manifest, but should some hyperacidity continue it may almost invariably be controlled by one or two lightly poached whites of eggs given shortly before the pain due to the hyperacidity usually commences.

These are the cases also in which purées of green vegetables and fruits may usually be introduced with advantage before starch foods. When starch food is introduced it is advisable at first to prescribe one entire meal of it each day, thus giving two meat meals (perhaps with green vegetables or fruit in addition) and one exclusive starch meal. Taken in this way the starch food will frequently be adequately digested, whereas a mixed proteid and starch meal might result only in imperfect digestion of both.

General Observations.

Hunger.—A short time after commencing the diet it frequently happens that patients—owing to the small bulk of the diet and the constant flushing with hot water—become very hungry and clamour for more food, although it may be found by fæcal examination that they are already taking as much at meals as they can adequately digest. If such is the case a few spoonfuls of minced meat or a cup of beef tea fortified with peptonoids, plasmon or sanatogen, may be given two and a half hours after each meal. If this disagrees, a small piece of unsweetened chocolate may be given instead. This is most effectual in assuaging the feeling of hunger and may usually be taken with impunity.

Sleeplessness occasioned by hunger may be controlled by similar means or by a teacupful of the beef tea mentioned above or beef jelly.

Fluid with meals.—Undoubtedly it is a good rule for all chronic dyspeptics—with the possible exception of cases of hyperchlorhydria—to eschew fluid with meals. This rule is easy of fulfilment for patients following a rigid proteid diet, thanks to the copious ingestion of water previous to meals, combined with the fact that an exclusive nitrogenous diet is not conducive to thirst. If patients cannot be weaned from the habit all at once a few sips of hot water or beef tea may be taken at the end of the meal. Provided there are no nervous or other obvious contra-indications, patients may also be allowed a cup of clear tea with the afternoon hot water, taking correspondingly less of the latter.

Loss of weight and weakness.—Loss of weight almost invariably ensues during the early stages of treatment, but need cause no anxiety, weight being quickly regained as soon as other foods can be introduced into the dietary, and, at times, even before this, for it not infrequently happens that patients whose condition necessitates a long course of the exclusive meat diet gain considerably in weight upon the meat alone.

Loss of weight is naturally accompanied by some weakness, but this, again, need cause no anxiety provided the diet is efficiently prepared, is being adequately digested, and the patient is taking sufficient rest.

It need hardly be mentioned, however, that the possible advent of these symptoms, as also their temporary character, should be carefully explained to the patient upon commencing treatment so that he may not feel discouraged if they arise. Discouragement, however, is seldom experienced, since before the weakness becomes apparent the general dyspeptic symptoms have usually greatly improved.

Rest.—From the preceding remarks it will be gathered that a certain amount of rest should be made essential during the early stages of treatment. Even patients whose strength is but slightly impaired should rest for at least half an hour before and after meals, while for patients who are very debilitated entire rest should be enforced for a few weeks.

Teeth.—Digressing somewhat from my subject, I would emphasise here the importance of making a *thorough* examination of the teeth in all dyspeptic conditions. I say "thorough," because, although septic stumps may not be present, a careful examination not infrequently reveals some degree of pyorrhœa alveolaris—a condition far too often overlooked—which, obviously, must be remedied if the dyspeptic condition is to be permanently benefited.

Additional Therapeutic Measures.

Although when the exclusive proteid diet is adopted it is necessarily regarded as the essential basis of treatment, it does not obviate additional therapeutic measures, which, applied with discretion, will greatly hasten recovery. These measures will naturally be applied in accordance with the necessities of individual cases, and I only presume to offer here a few general remarks concerning them.

Drugs.—It is frequently beneficial to give two to four drachms of phosphate of soda in the matutinal portion of hot water in order to cleanse the primæ viæ and obviate any tendency to constipation which the concentrated nature of the diet may occasion.

If the digestive processes are very feeble, ferments, such as ingluvin or papain, may be given for a brief period at the commencement of treatment. Taka-diastase may also be administered temporarily if starch digestion is troublesome.

Anti-fermentatives will frequently hasten the removal of abnormal fermentation, but should be given with care, since many are irritant in nature and have but little real value. Personally the anti-fermentatives I have found most useful are a combination of magnesium peroxide and poplar charcoal made up in the form of biscuits, and a mixture of beef peptonoids and creosote.

With regard to *tonics*, I believe it to be sound treatment to withhold them entirely for some weeks after commencement of the diet. Then, with invigorated powers of digestion and absorption, they will often prove most valuable, although, previously, the patient may have been saturated with the same drugs off and on for months, or even years, without appreciable benefit.

Massage and electricity.—In very debilitated cases where temporary confinement to bed is advisable, massage and faradism will often prove most helpful. In cases also of marked atony or dilatation of the stomach, faradism, or, even better, the triphase sinusoidal current with an intra-gastric electrode, is most valuable in hastening the reduction of the stomach to its normal capacity.

Lavage.—Owing to the copious ingestion of hot water and the small bulk and comminuted condition of the diet, lavage is seldom necessary except in extreme cases of dilatation. Where gastritis is a prominent feature, however, it is a good plan for the patient to massage over the region of the stomach for a few minutes after each quantity of hot water in order to expedite the removal of mucus from the gastric mucosa.

Hydrotherapy.—Of the various hydrotherapeutic measures which may be employed, "the Priestnitz Pack" is the simplest and probably the most beneficial, being particularly helpful where gastralgic pain is a prominent feature.

SUMMARY.

Briefly, the aim of the dietetic treatment I have endeavoured to describe may be summed up in two words, *elimination* and *re-construction*. In the first, or eliminative stage, the exclusive meat diet imposes but a minimum of work upon, and so rests, the digestive organs. At the same time, auto-toxæmia with all its pernicious influence tends to be effectually eliminated, proteid putrefaction being discouraged by careful quantitative regulation of the meat, acid fermentation inhibited by the absence of carbohydrate food. This process is assisted by the copious ingestion of hot water, which tends to eliminate toxic products which have already accumulated in the system.

In the second, or reconstructive, stage the patient is gradually built up upon a sound basis, the digestive organs, refreshed and invigorated by rest, cleansing, and the absence of toxic irritation, being now able to deal adequately with the additional food which is progressively introduced until a normal standard of diet is attained.

From the above it will be evident that the diet is pre-eminently suitable for those cases of long standing in which the digestive, assimilative, and metabolic processes are so perverted that the usual lines of treatment continuously fail to exercise more than a temporary influence.

Admittedly the *régime* is rather severe, and there is a certain proportion of patients whose feeble self-denial will prove unequal to the strain placed upon it. Such patients, however, are surprisingly few, since the diet, if properly prepared—and practically everything depends upon this—is very appetising, and they are encouraged by the rapid amelioration of symptoms which generally ensues. They are also encouraged to adhere to the diet by the periodical analyses of the excreta, particularly the examination of the fæces, the obvious common-

sense of which in deciding what foods they can and cannot digest appealing strongly to their intellect. Moreover, it renders those in whom "the spirit is willing, but the flesh is weak" less likely to transgress, since, as Strasburger (4) has truly observed, "The impression is never effaced if an error in diet is revealed to the patient from the examination of the fæces."

Preparation of the Diet.

The diet may be taken in the form of beef mince or beef cakes, the former being rather more digestible but less satisfying than the latter.

Both should be prepared from "topside" steak, preferably from the centre of the round of beef.

(A) *The beef mince.*—Take a steak, not less than an inch thick, free it from fat, place in a frying-pan —previously rubbed with a little butter to prevent sticking—and cook quickly (turning occasionally) over a clear, sharp fire until it is almost *half done.* Allow it to cool, then cut into strips and pass three or four times through an "Enterprise" mincer. Place the minced meat in an enamelled or aluminium saucepan, add a little water (about a teaspoonful to one ounce of meat is the average quantity) and beat to a smooth, stiff pulp with a wooden spoon; it must never be watery. Place the saucepan on an asbestos cooking-mat upon a cool part of the stove and cook slowly, stirring and turning over the mince constantly until the red colour disappears and it is thoroughly brown. This will take from fifteen to twenty minutes for a small quantity. If properly prepared at a gentle heat the mince will be quite smooth and of the consistency of soft dough. If it is granular it shows it has been cooked too quickly, and must be rejected.

I have found mince prepared in the way indicated from partially cooked meat to be easier to prepare, more appetising, and more easily digested than when prepared from raw steak as is usually advised.

(B) *The beef cakes.*—Pass strips of *raw* steak three or four times through the mincer. Place the minced meat on a plate, flatten out with a fork, and add a little water. By means of two forks quickly shape the meat into a round, flat cake about an inch thick, touching the meat as lightly as possible, just sufficient to make it hold together. Place the cake in an "Enterprise" griller, previously rubbed on both sides with a little butter. Hold the griller about eight inches above a sharp, clear fire, turning it about every ten seconds until the cake is cooked. The time required for cooking is seven or eight minutes.

Condiments, such as pepper, salt, and mustard may usually be allowed, unless hyperchlorhydria is a prominent feature.

Note.—It is essential to use the "Enterprise" mincer, this being constructed so as to strip the connective tissue from the meat during mincing. This connective tissue is retained on the inner side of the front disc of the machine and requires to be removed from time to time during the process of mincing.

REFERENCES.

(1) 'The Polyclinic,' October, 1906.

(2) 'The Relation of Alimentation and Disease,' 1895.

(3) "The Value of Fæcal Examination in Chronic Diseases," 'The Medical Annual,' 1908.

(4) "Diseases of the Digestive System," "Modern Clinical Medicine,' 1907, p. 611.

November 2nd, 1908.

A DEMONSTRATION OF CASES AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

By RAYMOND JOHNSON, F.R.C.S.

GENTLEMEN,—The first patient this afternoon is a man, æt. 40 years, who presents a strange combination of affections. His right foot, you will notice, is in a position of extreme equinus and the muscles of the calf are atrophied—a condition suggesting at first sight that the deformity has resulted from infantile paralysis. Further, the right knee-joint is moderately enlarged, and the movements, which are impaired, are accompanied by fine crepitation—a state of things suggestive of osteo-arthritis. Again, the little finger of the right hand is contracted, and it is seen that the contraction is diminished by flexing the wrist and greatly increased by extending it, so that we at once recognise that the contraction has a muscular or tendinous origin. Lastly, we

find on abdominal examination a large tumour, with ill-defined outlines, filling the left iliac and lumbar regions. The tumour is firm but indistinctly elastic, and the skin over the left half of the abdomen presents a brownish staining. Judging from the characters of the tumour alone, I should have regarded it as a large sarcoma, probably of the retro-peritoneal tissue. I may, however, at once tell you that we find a common explanation of all these different conditions in the fact that the patient is the subject of *hæmophilia*. Since his infancy the man has been observed by various physicians and surgeons, and has suffered from almost every variety of hæmorrhage. He has had bleeding from the gums and epistaxis, severe hæmorrhage after tooth extraction, several attacks of hæmaturia, hæmorrhages into the joints, and innumerable bruisings and subcutaneous bleedings. The deformity of the right foot is the result of hæmorrhages into the calf muscles, occurring chiefly in the years 1888 and 1893, the swelling on each occasion being accompanied by extensive discoloration of the skin. In 1893, whilst getting about on crutches, he struck the inner side of his right forearm in alighting from an omnibus; swelling and bruising followed, and gradually the contraction of the little finger supervened—a particularly unfortunate occurrence, as it prevented the man continuing his profession as an organist. The condition of the knee is another result of local hæmorrhage, and we know that a joint condition in such cases is not uncommon, which, in the absence of a knowledge of the fact that the patient is a bleeder, may lead us into serious errors of diagnosis.

The abdominal tumour is undoubtedly a large hæmatoma, probably in the retro-peritoneal tissue; it appeared rapidly about twelve weeks ago while the patient was suffering from a severe cough. At first the bruising of the skin extended upwards to the chest and downwards to the penis and scrotum. Five years ago there appeared a tumour in the same position which slowly subsided. I may add that the patient is unaware of any other case of hæmophilia in his family; his eldest brother, who died at thirty of consumption, and another brother older than himself, presented no unusual tendency to hæmorrhage.

The contraction of the finger in this case is particularly interesting, because last year Mr. Groves, of Bristol, recorded two cases of Volkmann's contracture occurring in hæmophilia. We are aware that this form of contraction of the wrist and fingers is sometimes met with after fractures of the arm and forearm, usually in young subjects, and no doubt results, in part at least, from damage to the muscles caused by the pressure of splints and bandages. In hæmophilia the damage to the muscles and their subsequent contracture is due to hæmorrhage into them. In both the cases described by Mr. Groves the most striking feature was inability to supinate the hand.

This little girl, æt. 3 years, is obviously the subject of well-marked *torticollis*. The head is drawn down towards the left shoulder, and is at the same time rotated so that the face is directed to the right. In the left side of the neck is a very tense band produced by the sternal part of the sterno-cleido-mastoid muscle. You will further notice that the child already presents quite distinctly the asymmetry of the face, which always shows itself sooner or later in these cases. The left side of the face is distinctly smaller and flatter than the right. On questioning the mother we find that she first noticed that the head was held on one side when the child was about sixteen months old, and we further learn that the child was born "the wrong way first," and that when it was about a week old it had a "gland" in the left side of the neck, which gradually got smaller and in three months had disappeared. We may thus regard the sequence of events to have been as follows: As the result of some difficulty in delivering the after-coming head the left sterno-mastoid muscle was bruised and perhaps torn, and a hæmatoma formed in it; this slowly absorbed, but gradually the damaged muscle underwent some degree of fibroid degeneration, leading to permanent contracture and wry-neck. It may perhaps be open to doubt whether in these cases the hæmatoma is itself the actual cause of the contracture, or whether the same injury to the muscles which causes the hæmatoma may not also cause a sort of ischæmic contracture of the muscle. Be this as it may, there can be no doubt that the usual cause of the infantile variety of wry-neck is injury to one sterno-mastoid muscle during birth. No treatment is called for in the case of an infant which presents one of these hæmatomas of the sterno-mastoid, but the mother should be told of the possibility of wry-neck supervening, so that she

may recognise it early. When once definite torticollis is present the shortened muscle should be freely divided by an open incision. After the operation the child should be kept lying flat on the back with the head fixed by sand-bags in an over-corrected position. In mild cases no instrumental treatment is required, but after a fortnight in bed regular massage of the neck muscles and passive movements and exercises are all that is necessary. In bad cases, however, it may be wise to adopt some method of maintaining the head in the desired position. One of the best methods is to apply a plaster-of-Paris jacket and a closely-fitting cap or a band of plaster-of-Paris round the head. The cap and the jacket are each provided with a number of loops of tape, and to one or another of these is attached a stout rubber cord, so arranged as to maintain the head in an over-corrected position; thus, if the left sterno-mastoid had been divided the rubber band would be attached to the right side of the head behind the ear and by its lower end to the front of the plaster jacket, so as to supplement the action of the right sterno-mastoid. The importance of dealing with these cases before the asymmetry of the face becomes noticeable is obvious.

This old woman, æt. 73 years, presents a diseased condition of the skin over nearly the whole of the dorsum of the right hand. The patch, which is well defined and extends on to the knuckles, is slightly raised and presents a nodular surface. It is in parts coarsely warty on the surface and in other parts is covered by small dry crusts, on removing which superficially excoriated surfaces are exposed. The affected skin moves freely on the parts beneath, but it is markedly thickened and there is clearly a new formation in its substance. The disease began as a small spot in the skin six years ago and has continued to spread slowly and painlessly. The case almost certainly is an example of a chronic form of *tuberculous disease of the skin* —the variety known as lupus verrucosus, which has not uncommonly been observed as one of the manifestations of senile tuberculosis. It may be noted that the patient presents no other evidences of tuberculous disease, but the gland above the internal condyle of the humerus is very slightly enlarged.

The patient's husband and two daughters died of "consumption," and I have tried to obtain evidence suggestive of a possible direct infection of the skin, which was very probably the mode of origin, but have failed. The husband and one daughter died some years before the patient first noticed the disease on her hand, and the other daughter was not attended to by her mother during her fatal illness. I may remind you that this form of tuberculous disease of the skin is closely allied to the so-called "anatomical tubercle" or "post-mortem wart," the tuberculous nature of which has been for some time recognised. In some cases of lupus verrucosus the papillomatous surface of the patch is so pronounced that the disease is mistaken for a papilloma. An interesting specimen in which this mistake was made is in the Museum of University College Hospital; it had for years been regarded as a papilloma of the skin of the foot, until microscopic examination showed that the disease was a typical tuberculosis of the skin. With regard to the treatment of this particular case I should advise excision of the affected area of the skin followed by grafting, but the patient is averse to any kind of operation, and at her advanced age I do not think it can be urged.

We have here the case of a woman, æt. 42 years, who complains that during the last four months her left knee has been swollen and painful. The onset was gradual, and there is no history of injury. On examination it is at once obvious that there is considerable swelling in the region of the knee, and the first point in the diagnosis is to decide whether the enlargement is due to disease of the joint itself or of one of the bones entering into it. It will be noticed that the greatest circumference of the limb is around the middle of the patella; this and other points to be referred to make it clear that the swelling is due to disease of the articulation itself. I mention this point because a most serious error in diagnosis may arise from mistaking a tumour of the lower end of the femur for disease of the knee-joint. On further examination we find that the enlargement of the knee seems to be due chiefly to thickening of the synovial membrane, which can be appreciated most easily in the supra-patellar pouch, where the upper limit of the synovial membrane is felt as a firm, rounded and somewhat nodular border.

It is not possible to demonstrate the presence of excess of fluid in the joint, at any rate the patellar tap cannot be obtained. The movement of the joint is limited, but such as is present is not accom-

panied by crepitation. When the limb is grasped with the two hands above and below the knee it is not possible to obtain any unusual amount of lateral mobility in the joint, thus showing that the ligamentous structures of the joint are not relaxed. Finally, over the lower border of the patella and the patellar ligament is a well-marked, apparently solid swelling, due almost certainly to thickening of the wall of the prepatellar bursa. There is no evidence of any other joint being affected. What is the nature of this monarticular joint affection? It does not seem at all likely that it is rheumatoid arthritis, for amongst several objections to this diagnosis is the absence of lipping of the edges of the articular surfaces and the smoothness of such movement as is possible. Nor is there any evidence to suggest that the disease is of neuropathic origin. In any case of chronic joint affection it is always well to bear in mind the possibility of a septic origin—a chronic septic arthritis—such as may closely simulate other forms of chronic joint affection, and is found in association with some source of chronic septic infection. In this case, however, there is no evidence of such a cause. Taking into account the fact that the most striking feature of this enlarged joint is the very marked thickening of the synovial membrane, one naturally thinks at once of tuberculous disease as a very probable diagnosis. The very definite enlargement of the patellar bursa must not be overlooked, and should, I think, suggest the possibility of the syphilitic nature of the disease. Although tuberculous disease of bursæ is well known, I think I am right in saying that tuberculous disease of the prepatellar bursa is very rare. On the other hand, syphilitic disease of bursa is not uncommon, and especially tends to affect this particular bursa, causing, as in this case, an enlargement which is chiefly, if not wholly, solid. At any rate I feel sure we shall be right in trying the effect of a course of iodide of potassium.

I should mention one important fact which the patient has told us. Several years ago she was under treatment for "stricture of the rectum and fistula." Some kind of operation was performed, and the question of performing colotomy seems to have been considered by a very competent surgeon. I have not yet made a rectal examination, but it is more than probable that the condition was syphilitic in nature.

The last case is a very ordinary one, a child, æt. $4\frac{1}{2}$ years, with *tuberculous disease of the spine*. The mother says that about eighteen months ago the child had a fall and complained of pain in the back. Nothing further happened until five or six months ago, when it was noticed that the child's back was "growing out," and a few days ago the right buttock was first observed to be swollen. The child is pale and ill nourished, and there is a typical kyphosis in the lower dorsal region. The enlargement of the right buttock is quite obvious and is clearly due to a fluid swelling beneath the gluteus maximus—undoubtedly a chronic abscess. In feeling for fluctuation in the buttock it is very necessary to palpate in the direction of the fibres of the muscle and not across their length, for we thus avoid the fallacy of suspecting fluid when none is present, on account of the sense of fluctuation given by a muscle when palpated with the two hands in a direction at right angles to the line of its fibres. The abscess in the buttock has probably found its way out of the pelvis through the great sciatic foramen. Further examination also leads to the detection of a deep-seated elastic swelling in the right iliac fossa, and when firm pressure is applied to it a slight but unmistakable sense of fluctuation is felt just below Poupart's ligament outside the femoral artery. So that there is clearly an abscess coming down in the right psoas muscle and just beginning to extend to the thigh. The child has complete power in its lower limbs, normal reflexes and sensation, and complete control over the bladder and rectum, so that there is no evidence of any pressure on the spinal cord. When the disease of the spine was first detected a plaster-of-Paris jacket was applied, and the child was allowed to get about. Now, valuable as is the use of a plaster jacket in certain stages of spinal disease, I do not think it can be regarded as efficient in the early active stage. It is essential at this period, whether the patient be a child or an adult, that he should be kept in the recumbent position. If the patient is a child it is useless to try and keep it in the recumbent position, at any rate in the circumstances of the hospital outpatient, without the use of some kind of apparatus. A most uniformly useful appliance is a double Thomas's splint with a head support. This splint can be adapted to suit cases of disease in any region of the spine—the child can easily be carried about without harm—and the splint is equally applicable if a spinal abscess has required operation. It may safely be laid down as a general rule that a child with tuberculous disease of the spine should be kept in the recumbent position for at least a year, and that in whatever position the disease is situated the child should be in an apparatus which extends from the head to the feet.

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WITH DR. BRADFORD IN THE WARDS OF UNIVERSITY COLLEGE HOSPITAL.

THIS patient is a married woman, æt. 26 years. About a week ago she had her tonsils removed. Previously to that she had had flying pains about the joints, and these became infinitely worse after the tonsils had been removed, and she had to take to her bed, where she has remained since suffering from these painful and swollen joints. The joints involved were the left knee and both wrists; she has also had pains in her shoulders and in one ankle. It is the first actual attack she has had. But there have been odd pains five weeks previously to this, and she also has had a vaginal discharge. The wrists are a little enlarged, and she says she often has sore throats. She has two children, the youngest being three years old. She has not recently had any miscarriage. The left knee was involved, but there is no sign of the affection of that joint now. There is no morbus cordis. Now, what is the diagnotic point about this case? The sore throat and the vaginal discharge are the two essential facts. With regard to the warning pains in her limbs, I think we may put those down to the pyrexia and the local trouble. It may be acute rheumatism, or it may be arthritis due to septic infection. This is a rather interesting type of case, because it is the kind which has led some people to say there is no such disease as acute rheumatism, that there is not a definite morbid process due to a single cause and running a definite course known as acute rheumatism, because you see cases simulating acute rheumatism in the matter of joint lesions where there is a possibility of septic infection. Therefore that is the diagnostic question in such a case as this: Is it acute rheumatism or is it one of some kind of septic arthritis associated with the vaginal dis-

charge? You know that arthritis, as distinguished from acute rheumatism, may be due to a very great number of different poisons; you may have a virulent condition producing a suppurative arthritis, such as occurs in pyæmia, or you may have at the opposite end of the scale what one talks about as acute osteo-arthritis associated with some uterine or pelvic lesion, or perhaps sometimes associated with some chronic pulmonary lesion such as bronchiectasis, or long-continued chronic bronchitis, at any rate some condition in which there is a septic lesion and absorption has taken place from it. There are all grades of severity, from the most virulent septic arthritis, which destroys the joint in a few days, to chronic osteo-arthritis, which goes on for months, or even years. And about half way in that series you may put gonorrhœal rheumatism, as it is called. That sometimes resembles in severity septic arthritis, while in other cases it runs a chronic course, so that it more nearly resembles osteo-arthritis. The point I want to draw your attention to is, that where there is a definitely well-known source of septic infection, whether it be gonococcus or some other form of microbe, you have a whole range of joint troubles which are capable of being produced, in the one case of a suppurative type, and in another case of a chronic progressive type and of a destructive character. In the one case there will be severe inflammatory disturbance, while in the other there may be nothing more than a thickening of the fibrous tissue of the capsule around the joint. There are many degenerative changes, such as erosion of cartilages, the wearing away of bones and so on, but the only inflammatory change will be found in a thickening of the capsule and the formation of fibrous tissue. The fact, then, that in some cases there is a joint effusion, not necessarily purulent, has led to the extreme idea that there is no such disease as acute rheumatism, and that the latter is only a clinical label attached to a particular group of cases of arthritis of unknown ætiology. I may say at once that I do not believe it to be true for a moment. I think there is such a disease as acute rheumatism, but there is a modern school of bacteriologists who, in the light of the great difficulty there has been in finding the organism of acute rheumatism, and the conflict of opinion as to whether there is such an organism, and whether it has been definitely found at the present time or not, hold that an acute rheumatism is nothing more or less than one of those forms of septic arthritis. As regards the clinical differences, I do not think you can tell from the appearance of the joints. There are many cases of septic arthritis, and of acute osteo-arthritis, where the appearance of the joints is strikingly like that of acute rheumatism, where there is swelling with considerable effusion and great pain, and there is no evidence of bone destruction and no evidence of erosion of cartilages, so that the superficial appearance of the joints simulates very closely that found in acute rheumatism. I think that is so here. You cannot tell, from looking at that wrist joint, whether it is rheumatism or not. Notice the blush over it. That is like a rheumatic joint, but you get that also in some cases of pyæmia and septic joints. To settle the question at once, what is the main clinical reason for thinking there is such a disease as acute rheumatism? That it runs a definite course. That is the most conclusive argument. Of course the modern generation does not see it run its full course—we cut it short. If left alone that disease will run a definite course of nineteen days. That is an absolute characteristic of a specific infection or disease. But nobody can say how long an inflammatory disease will run. If there is a pyæmic joint you do not know how long it will last. If there is what is called gonorrhœal rheumatism—a very bad name by the way, because there is no rheumatism about it—you do not know how long it will continue; it may be a few days, or months, or even years. But rheumatic fever has a definite duration. The other reason is that you can cut it short with salicylates. But a clinical difficulty comes in in this way, that every now and then you come across a case which you think, and so does everybody else who sees it, is rheumatic. It looks like a rheumatic case, and you cannot trace any septic infection, but when you give salicylates it does not clear up. You will all see a case of that sort sooner or later, and the question is what are these cases. Those are the cases which the bacteriologist, as distinguished from the clinician, would appeal to to prove that there is no such thing as acute rheumatism. But many of these cases which do not clear up under the influence of salicylates are found to have an inflammatory complication, such as endocarditis or pleurisy for

instance. That accounts for one group of puzzling cases. And then there are other cases which are erroneously diagnosed. They are really not cases of rheumatism at all, but of arthritis due to some other cause. A good illustration of that is the joint lesion in the convalescence from pneumonia, which may simulate acute rheumatism. And you may see the same thing possibly in scarlet fever. But the case of scarlet fever is a little more complex. The arthritis of scarlet fever, which is often spoken of as scarlatinal rheumatism, is perhaps an arthritis due to some other cause. So in one's own mind there is no doubt about the existence of such a disease as acute rheumatism. There are cases of septic arthritis of mild type which mimic or simulate it very closely, and I think you could not say here at first which condition is present. Her history of repeated sore throats is in favour of rheumatism, because acute rheumatism often manifests itself by tonsillitis recurring. The joint lesion is not always the initial condition. You often find tonsillitis first, and it is well to bear that in mind in the case of children who come under observation with morbus cordis, but who have no definite history of rheumatism. This patient's age is 26. She is a little old for a first attack of rheumatism, because it is a disease which you usually see at a younger period of life. Still, it is within the limit of age for the onset of acute rheumatism. But when you get a similar appearance in a patient ten years older than this woman, you should be very sceptical about it being really rheumatism. How shall we know ultimately whether it is rheumatic or not? We shall know partly by the result of the administration of salicylates. She only came in last night. We spoke of rheumatic fever running a definite course, now we have mentioned the effect of salicylates, and there is a third means of ascertaining what it is. If you take pneumonia, diphtheria and rheumatism as three illustrations, they are all diseases in which during the height of the malady you have a profound change in the structure of some part of the body, most marked in the case of pneumonia because the lung becomes solid. It is fairly well marked in diphtheria, with the inflammation, etc., in the throat, and in the case of rheumatism there are the changes in the joints. They are all three remarkable for the fact that, however severe may be the illness, it leaves nothing behind it of a permanent nature. It is not absolutely true that in no case of pneumonia is the lung damaged by the pneumonic process. Sometimes there are pulmonary complications, but broadly my statement is true. The same thing is true of rheumatic joints. Notwithstanding the severity of the effusion the pain and the discomfort, the thing clears up, the patient is not left with a stiff joint, and there is no impairment of the subsequent mobility of the joint. That is very characteristic of rheumatism. And if you see a patient who tells you he has had acute rheumatism and the joints are crippled, be very sceptical about it being rheumatism. Most of those cases which look like that are some other form of arthritis, due to some septic process. Personally, I am very sceptical about the existence of the disease sometimes called rheumatic arthritis. You will see described in text-books acute rheumatism, osteo-arthritis, and sometimes sandwiched in between the two an affection which is spoken of as rheumatic arthritis, by which is meant a disease which begins like rheumatism and has a clinical course somewhat like rheumatism, except that it is more prolonged, which does not respond to salicylates, and which differs in the fact that the patient's joints are more or less stiff afterwards. I think many of those cases are arthritis due to some other infection or some other organism. So we shall be able to know by the influence of salicylates as to whether it subsides completely under them, and that it is the best evidence as to whether it is rheumatic or whether it is due to other trouble. There is pain in this wrist when she moves it. If the pain is due to rheumatism it should disappear in twenty-four hours under the influence of salicylates, and the effusion ought to go in less than forty-eight hours. And the pyrexia should come down in two stages. It is a very interesting thing about rheumatism that you will not bring down the temperature of rheumatic fever by giving salicylates at once in whatever doses you may administer it. It will first come down to about 100° F., and it will remain somewhere about 100° for two or three days, and then it will come down to normal. So if you have a patient whose temperature is 104° F., or even higher, in twenty-four hours under the influence of salicylates it will come down to 100°, but it will not be normal until the best part of a week has elapsed. So we shall know by the subsidence of the pain and the coming down

of the temperature and the clearing up of the local lesion in a very short time whether it is rheumatic or not. The temperature chart leads me think it is rheumatic.

Now, does the observation of the joint affected help in the diagnosis as to whether it is acute rheumatism or not? It does not help you very much. The ankles and the wrists are those most likely to be affected in a case of acute rheumatism. It not uncommonly begins in the ankle, but it is not uncommon for them to be affected in septic arthritis of the type I have been speaking of. Acute osteo-arthritis is an important malady for you to be able to recognise. As I have said, I do not think this patient is a case of it; I think it is acute rheumatism. Osteo-arthritis is very prone to involve the wrists, and to occur in a young woman like this, and to occur after confinement, in association with some chronic uterine discharge. It is a very serious malady, because it produces a great degree of joint trouble and in a very short time, and it does not respond, in the way rheumatism does, to salicylates and to ordinary methods of treatment. Sometimes in the course of a very short time you may get almost all the joints of the body involved. One set of joints more particularly apt to be involved and to produce very characteristic symptoms is that between the vertebræ. These articulations are very rarely affected in acute rheumatism, but in acute osteo-arthritis they are often affected, and sometimes in chronic osteo-arthritis. As you may well understand, it produces great pain and great deformity in those joints. The same remark applies to the jaw joint. The jaw is not liable to be involved in acute rheumatism, but it is liable to be affected in acute osteo-arthritis. With regard to the salicylates, in hospital practice it is best to give them in small doses at short intervals, because you have the patient fully under control and you can produce the effect of the salicylates very quickly. And you should remember that the salicylates relieve the pain first, and then the fever, and that the fever is relieved in two stages. A very curious thing about the action of salicylates in rheumatism, which has never been really satisfactorily explained, is that, although they control the arthritis, they do not control to the same extent the pericarditis or the endocarditis. The cut and dried reason usually given as to this is that the joint is kept at rest, whereas the heart does not rest. But you must remember that the action of salicylates in rheumatism is perhaps different from the action of quinine in malaria and of mercury in syphilis, There are some reasons for thinking that the action of salicylates is not altogether a specific one; if it were so one does not see why it should not control the endocarditis.

There was a patient here a little time ago who had recurrent tonsillitis. The tonsil was removed, and next day he had an attack of rheumatic fever. There is no doubt many cases of tonsillitis are really cases of rheumatic infection, and it is very important to bear that in mind in the case of children. You will so often see cases of children with morbus cordis where there is no obvious history of rheumatic fever. When you have cases of tonsillitis in children you should keep the patients in bed so as to rest their hearts as much as possible, always bearing in mind the possibility of the condition being rheumatic. And that may also explain how not uncommonly in tonsillitis the condition can be controlled by salicylates.

This patient is a married woman, æt. 48 years. She has had, on and off for eighteen months, symptoms of shortness of breath and palpitation, with cough and nocturnal starts. Latterly her feet and ankles have begun to swell, and she has also had swelling at the back of the sacrum. Her urine has been diminishing in amount. She has never had actual rheumatic fever, but she has had chronic rheumatism, and there is a history of one miscarriage. There is a strong history of heart trouble in the family. She has not had any hæmoptysis. She was never troubled with her heart until eighteen months ago. She has had pain in the præcordium. Notice she gets short of breath even if she moves much in bed. The œdema in the legs is of only recent onset. There is no ascites. Her main symptoms, then, are shortness of breath and cough with pain. She says she has always had pain in her heart. One can feel the pulsation of the heart over a very large area. The history which we have been given is rather that of mitral regurgitation than of mitral stenosis. You would not expect a case of mitral stenosis to present urgent symptoms like this without having in the past history the occurrence of hæmoptysis. That you nearly always have in such a case, because the degree of embarrassment of the

pulmonary circulation is so great. Directly you feel this patient's pulse you know it is that of mitral regurgitation rather than of mitral stenosis, because of the extreme irregularity. Patients with mitral stenosis do not have an irregular pulse, whereas patients with mitral regurgitation commonly do. The irregularity is not very obvious in the præcordium, in other words, it is much more obvious at the wrist. This is a case in which you get quite an erroneous opinion as regards the rate of the cardiac rhythm by taking the pulse—the heart is going much more quickly than the pulse. The next thing to look for is the state of the veins in the neck. You know from the old statement that if you want to gauge the condition of the left ventricle you should feel the pulse, and if you want to gauge the condition of the right ventricle look at the veins of the neck. They are distended, you see. Another thing to look for at once is the presence, and if present the degree, of hepatic enlargement. There is certain to be some enlargement of the liver. The liver is distinctly tender, so you can tell at once that you are dealing with an acutely congested organ—a point of some importance with regard to prognosis, and also to some extent with regard to treatment. One of the most important things in regard to mitral disease is to bear in mind that although you get venous distension in mitral disease you do not get it uniformly, and that the distress and the back effects are spent on the hepatic circulation. Here the organ reaches nearly to the umbilicus and is tender, painful on pressure, and yet she scarely has any œdema of the legs. And what œdema of the legs there is might very well be attributed to another cause, *i. e.* varicose veins. Whether it is due to varicose veins or due to the cardiac condition, the œdema of the legs is small in amount, whereas the hepatic enlargement is considerable. You should always carefully examine the liver, and you will notice that I went through the examination of that before I listened to her heart. You will not learn very much about the condition of this patient's heart by auscultation; you will learn much more by the simple examination which I have made: the character of the impulse, the state of the pulse, the degree of distension of the veins in the neck, and the size and degree of tenderness of the liver. These will give you a better idea of the heart than auscultatory phenomena will. That is especially for the reason that when these patients come into hospital like this there has generally been what is called failure of compensation, cardiac breakdown, and the contraction of the ventricle is feeble and murmurs are very often inaudible. They are audible here. But many of these patients have no murmur when they come into hospital—they only develop a murmur as they improve. But the palpation phenomena, the character of the impulse, the degree of distension of the veins of the neck, the state of the pulse, the size of the liver, are things which will not mislead you. Another thing is the state of the urine. The condition of the urinary secretion gives you very accurate information as regards the efficiency of the circulation. That you test mainly by the quantity of urine that the patient secretes, by the specific gravity, and also by its colour. All those things give you a fairly accurate information as to whether the functions of the kidney are being satisfactorily performed, and therefore whether the circulation through the organ is fairly efficient. The mere question whether there is albumen, or how much there is, is of comparatively less importance. The urine is very dark in colour and the specific gravity 1022. The typical cardiac urine is high coloured and loaded with urates, the kidney is not working well as a filter owing to the failure of the circulation, and it contains a cloud of albumen. So there is probably no very serious renal disease. Is there stenosis? That is a very much more difficult question to settle. The way in which one tries to settle it is, first of all whether there is a presystolic murmur. You must remember that a presystolic murmur may be present without there being any stenosis. There may be a well-marked presystolic murmur in cases where there is adherent pericardium and where the ventricle is kept dilated by that pericardium. So that even the presence of a presystolic murmur is not proof of the existence of mitral stenosis. The real difficulty is when the disease is severe the murmur may not be present. Therefore, how can you diagnose it? There are two things which lead one to suspect it. One is a shortening of the first sound. In this case you hear that the systolic murmur is only present with the strongest beats, and that in between the systolic murmurs and the stronger beats you may hear a shortened first sound, which is very like the second sound in character. Again,

you must look for accentuation and duplication of the pulmonary second sound. There is marked accentuation of the second sound, and sometimes it is reduplicated. So it is very probable that there is a double mitral lesion. But the essential lesion is regurgitation. We are putting her upon digitalis.

This woman is the subject of acromegaly, and we think there is trigeminal neuralgia on the left side. The question is, what is to be done for the relief of that neuralgia? She says it is as bad as ever, but it does not last continually, but goes away and comes back. The most efficient medicinal treatment for trigeminal neuralgia is a long course of arsenic in between the attacks, and for the relief of the pain caffeine and croton chloral is useful sometimes, but mainly in the spurious form, that associated with defects in the teeth. Gelsemium is useful sometimes, and on the whole caffeine, and gelsemium are the most useful. But where the disease is severe you are practically often obliged to use morphia. The hands have been X-rayed, and there is a little formation of new bone in the terminal phalanges of the thumb. Notice the size of the tongue.

This patient, a woman, æt. 64 years, is the subject of diabetes. On a full diet for three days she had 9½ oz. of sugar, 10·3 oz., 8·9 oz. in the urine daily. When dieted the quantity immediately fell to 2·6 oz., next day it was 3 oz., the next 2·4 oz. So the sugar was less than half what it was previously. I should have thought that in a case like this it would have been reduced still more. In treating diabetics with these patent foods it is well to test them, not to accept the foods supplied too readily, because some of them contain perceptible quantities of starch. When you can get the sugar down to this quantity by dieting it is almost certain that a large part of the remaining sugar is due to the fact that she has not been dieted rigidly enough. The outlook in such a case as this, where the response is so good, is very fairly good, particularly in a patient of this age. And these patients where the amount of sugar is greatly reduced like this will commonly tolerate the diet, because there is such a diminution in the urgency of the symptoms. The cases where it is not worth while to insist on dieting are those in which the diminution of sugar is very slight, and it is possible that in those cases you may do harm by having too rigid a diet.

In this patient there is a certain amount of œdema over the sacrum, and in the urine there is a large cloud of albumen. There was a tricuspid murmur, but it has recently disappeared. There has been no response to digitalis or strophanthus. There is a faint mitral murmur to be heard. With regard to the relative value of strophanthus and digitalis, personally I think that digitalis is very much the better drug. Strophanthus was introduced because it was said to slow the heart and increase the force of the heart beats while it did not contract the arterioles. It was a theoretical consideration that a drug which slowed the heart and did not contract the arterioles would be more useful in cardiac disease than one which did slow the heart with increase of force and contracted the arterioles. I think that theoretically the statement is wrong, because I think you should look at the circulation, not as caused by the heart but as caused by the blood-pressure. That is the proper way of looking at the physiology of the circulation; the heart is the apparatus for getting up blood-pressure, and the heart cannot do that unless there is a certain amount of peripheral resistance. You can see the truth of that statement by considering ordinary cases of fainting. The bulk of them are not cardiac but vaso-motor, and due to dilatation of the vessels of the abdominal area. It is a very unusual thing in ordinary syncope for the heart to be affected, except as a result of the great dilatation of blood-vessels. If you feel the pulse of a person who has fainted you will find that it is quick and feeble, and the feebleness of it is due very largely to the fact that the vessels are greatly dilated. If you look upon blood-pressure as the cause of the circulation, I think you will see that in treatment it may very well come about that a drug which raises the blood-pressure in a physiological manner by slowing the heart and constricting the vessels is a more sound and physiological method than giving a drug which produces its effects by acting solely at one end, namely, on the heart. It is a purely theoretical consideration and I do not think it should influence you. I think those writers who have advocated strophanthus on theoretical grounds are wrong. Every practical physician will tell you that digitalis is very much

more successful in the treatment of morbus cordis than is strophanthus. Strophanthus is highly toxic; it may cause a great deal of gastric irritation, far more than digitalis, and it is by no means such an efficient substance. But it is true that every now and then you may see a person who does not respond very well to digitalis but who will respond to strophanthus, but it is a rare event. Personally, I have given up strophanthus. At one time I used it a good deal, but I do not now employ it. And I think the infusion of digitalis is a better preparation than the tincture. The infusion of digitalis, like all other infusions, is of no use unless it is freshly prepared. It is often made of stale leaves, and for that reason will not be efficient. But if you get a strong and properly made extract, you will find it is far more potent and far less toxic than the tincture. And some of the toxic principles of digitalis are more soluble in alcohol than in water. I am sure that if you want to get the best results in the treatment of mitral disease it is best to use the freshly prepared infusion of digitalis. It is like the case of the extract of male fern. A man had a tapeworm, and went the round of the physicians in London but got no relief. He then went to a certain physician in Berlin who had a great reputation for treatment of such cases, and he was perfectly cured by him. And the whole point was that the Berlin physician took care to see that the extract was a fresh one, which there is reason to believe was not done in London.

This man, æt. 32 years, is another case of diabetes, and in him the response to diabetic treatment is not so great as in the woman we have just seen. I am told that the urine gives both the diacetic reaction and the acetone reaction, and the question is what stress is to be laid on these facts? I think I told you before that the only stress to be laid on it is that it shows the case is one of the more severe forms of diabetes, the so-called composite variety. I do not think you should look upon it as necessarily a premonitory sign of the occurrence of acetonæmia. It is a condition which does occur before acetonæmia, but it often occurs for months and even longer without any such complication. A much more important premonitory sign of acetonæmia is a diminution in the quantity of urine, and that is one which should make you careful. Another thing is the occurrence of inveterate constipation, and sometimes the occurrence of unexplained abdominal pain. All those things may be premonitory signs of acetonæmia. But I do not think the presence of diacetic acid or of acetone is a thing which should make you think that that complication is particularly prone to ensue.

This man is probably a case of vesical growth; he is suffering from hæmaturia. Renal hæmorrhage often occurs in granular kidney, and may take place from the mucous membrane of the pelvis of the kidney. The patient may lose considerable quantities of blood, comparable to that seen in association with malignant disease; but there are not clots, the blood is uniformly mixed with the urine. If there are blood-clots in the urine it commonly suggests the presence of a growth either in the bladder or in the kidney. Sometimes clots are passed in a case of stone, when there is a sudden or profuse hæmorrhage. This man was cystoscoped, and it is probable he has a growth in his bladder. These vesical growths in elderly men run a very chronic course. The villous ones may be associated with very profuse hæmorrhage and cause a high state of anæmia, but those which are dependent on such a serious thing as epithelioma may run an exceedingly chronic course, and if the patient does not have pain he suffers comparatively little inconvenience, except from hæmorrhage which recurs from time to time. I have known cases of that kind where hæmaturia has been present many years. I think the most satisfactory treatment in these cases if they cannot be removed is by means of nitrate of silver injections. I do not think it is of much use to give ordinary hæmostatic drugs. There is no indication for any active treatment here because he has not any hæmorrhage just now.

This patient, I think, has degeneration of the cardiac walls. I do not know the ætiology of it, and there seems to be no certain history to account for it. He has the physical signs of dilated heart and there is considerable arterial degeneration. His most urgent clinical symptom is dyspnœa, and that is very often the case with these patients who have cardiac degeneration. Very often they have no extensive dropsy, but this man has had dropsy, though not to a marked degree. And he has Cheyne-Stokes' respiration, which is mainly present

at night during sleep. I have already pointed out to you that the occurrence of Cheyne-Stokes' respiration during sleep is a matter of no great significance, but when it occurs by day when the patient is awake it has a pathological significance. The condition which this man has is one of those in which you are peculiarly liable to have Cheyne-Stokes' respiration, but it may occur in the terminal stages of almost any disease, but especially in respiratory affections. Putting these terminal conditions on one side, Cheyne-Stokes' breathing occurs mainly in cardiac cases, renal cases, and cerebral cases. There are some people who think that Cheyne-Stokes' breathing, even in cardiac cases, is of renal origin, but this man has no renal lesion. The condition was originally described by Stokes in cases of fatty heart. I do not think there is any reason for believing that the phenomenon is always of renal origin. Still, it is very characteristic of renal disease. What are the characteristics of the phenomenon? It is a variety of periodic respiration as distinguished from rhythmical respiration. The normal respiration is rhythmical, whereas Cheyne-Stokes' respiration is a grouped respiration. It begins gradually and fades away gradually. It is a mistake to look upon this breathing as a mere disturbance of the respiratory centre. There are certain changes in the pulse: it quickens up and slows down, and the quickening of the pulse and the slowing of the pulse is not perfectly synchronous with the respiratory disturbance. The pulse quickens just before the patient begins to breathe deeply, so that if you put your finger on the wrist and do not look at the respirations you can tell by the quickening of the pulse that the deep respirations are going to take place. If you want to be perfectly accurate you must not say that the respiration in one phase of Cheyne-Stokes' breathing ceases; as a matter of fact, the movements become so slight as to be imperceptible to ordinary methods of observation, but if you had a finger on the under-surface of the diaphragm you could feel it move, though but very little, and that it gets greater and greater in amplitude, and the pulse begins to quicken before the respirations begin to get violent. And then the pupil changes also, that is to say, it dilates and then contracts. It is a most striking thing to see for the first time, because the pupil dilates suddenly and widely just before the patient begins to breathe deeply, and when the period of deep breathing is passing away the pupil contracts. But the most interesting feature of all is the alternations in the degree of consciousness. In the occurrence of pathological Cheyne-Stokes you find that the patient is often only conscious during the period of dyspnœa, and that he is unconscious during the period of apnœa. And you can test that in a very marked way, if you like, by asking your patient a question in the dyspnœic period and again in the apnœic period. He will not reply in the apnœic period, but he will reply in the other. There is another feature of great interest in this condition, namely, the waxing and waning of activity of the motor centres. Look at the first case of cerebral hæmorrhage which comes before you, particularly somebody who is brought in unconscious, and you may not be able to see on which side the hemiplegia is present. But if the patient has Cheyne-Stokes' respiration he will move the non-paralysed side and generally carry out scratching movements on the front of the chest. In the ordinary non-paralysed patient, the cardiac case for example, or the renal case, you notice that there are general restless movements, and people watching these movements think that they are due to the patient, being uncomfortable and simply restless, throwing himself about. But it is not so, as shown by the patients who have a cerebral lesion which produces unconsciousness. You see the same waxing and waning of the motor centres which are still capable of acting. So, without going into details, I may say that in Cheyne-Stokes' respiration there is not only periodicity of the respiration but periodicity of the heart rhythm and of the pupil, and of the activity of the motor centres, and there is also a periodicity in consciousness. Thus you see the symptom is very complex. You are very prone to put those occurrences down to the fact that the patient is uncomfortable, but they are not really signs of distress at all, and they may occur when he is asleep, or still more important, when he is in a state of profound unconsciousness. In many cerebral cases you do not get the respiratory disturbance of the true Cheyne-Stokes' type, you are much more prone to get periods of apnœa and dyspnœa beginning and ending suddenly, the first breath he takes being very deep instead of being very shallow. You must have seen it in tuberculous meningitis. It is very important because it is sometimes the first thing which enables you to say for certain that a child has tuberculous meningitis. Many times one has been at the bedside of a child who has had headache and vomiting, and there has been doubt, but suddenly periodic respiration occurs and then there is certainty that there is a cerebral lesion. With regard to the waxing and waning of the activity of the motor centres it is most useful, because it enables you to tell whether there is a cerebral lesion, and enables you to tell on which side of the brain the lesion is, which is not always an easy matter if the patient is unconscious, but a person having Cheyne-Stokes' respiration does not move the paralysed limb.

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THE PROGNOSIS IN TUBERCULAR AFFECTIONS OF THE URINARY TRACT.

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Surgeon to St. Bartholomew's Hospital.

GENTLEMEN,—The subject which I propose to take for my clinical lecture to-day is tubercular disease as it affects the urinary tract, and I do so, first, because I have several cases of this affection in the wards at the present time, and secondly, because the prognosis of this condition varies considerably in different cases. This variation I hope to be able to prove to you is largely dependent on the way in which the urinary tract is invaded. If I am to lay before you facts which will illustrate the element of prognosis, it is obvious that I must adduce these facts from cases which have been under my care in times past and are now in excellent health, all symptoms of their trouble having completely subsided—I hope I may say vanished—for some years past.

I will begin by relating to you the history of a lady whose kidney was thus affected and removed by me some eighteen years ago. I may further tell you that since that time I have operated on her for piles and removed both ovaries as well as her uterus, which was the seat of one of the largest fibroids I have ever seen, and she is now alive and enjoys excellent health, so I think I may fairly claim that she at least believes that the removal of her kidney was a success. The history of her tubercular trouble was as follows. She is now æt. 47 years. When she was a girl she suffered with tubercular glands in the neck, which, according to the fashion of the day, were allowed to break down and suppurate, and have left ample scars to testify to their former existence.

For more than twenty years she had no further tubercular trouble, but when she was thirty years of age her kidney mischief started and this was how it began. Frequency of micturition was the first symptom of which she complained, with the occasional passage of thick urine. As this condition did not yield to ordinary remedies I was asked to see her. I examined her under an anæsthetic and introduced a cystoscope into her bladder. The principal part of the interior of the bladder was healthy, but round the orifice of the left ureter was a zone of redness and congestion, and it was evident that the thick urine came from that ureter only. Though the patient was rather thin and the left kidney region could be easily palpated and examined, there was no evidence of any alteration in the size or consistency of that kidney. I expressed an opinion that the left kidney was at fault, but so far as I remember I did not go so far as to diagnose tubercle with certainty at that stage of her disease. I suggested the kidney should be explored, but my patient was unwilling to accede to the suggestion and waited some weeks, during which, in spite of rest in bed and general medical treatment, she got steadily worse. When I saw her a few weeks later there was no doubt about the left kidney being affected. It had become painful, and was somewhat enlarged and tender to the touch. Feverish attacks had supervened, and the temperature was at times raised to 104° F. and over. An operation was urgently called for if her life was to be saved.

I operated and removed the kidney but left the ureter behind, contenting myself with cauterising and closing its upper end. It was not customary then to follow the ureter down towards the bladder and remove it freely, a course which would probably be pursued to-day. After a somewhat protracted convalescence complete recovery took place and there has been no evidence of any tubercular trouble since.

Let me relate another case. A man, æt. 27 years, was admitted into this hospital under my care in 1895—that is, thirteen years ago. He was passing pus with his urine, and his pain and general symptoms left no doubt that his left kidney was affected. His temperature was raised considerably above the normal, and he stated he had lost flesh during the previous two months to the extent of more than a stone. His illness began with some pain and uneasiness in the left lumbar region, and a week or so later the pain radiated into his groin and down the back of his leg. He was obliged to give up his work as a labourer within a fortnight of the onset of his symptoms. He had enjoyed excellent health up to the beginning of his illness and there was no evidence of any previous manifestation of tubercular disease. I want to emphasise this statement, but I also want to point out to you at the same time that tubercular lesions may be present in great abundance without giving any external evidence of their existence. One illustra-

tion will make this point clear to you. You will some of you recall a patient on whom I operated for ruptured gastric ulcer just over two months ago, in whom at the time of operation I found masses of calcareous glands of tubercular origin in the lesser omentum, but we failed to get any history of previous illness which could enable us to infer the particular period of her life when the tubercular infection occurred. Unless her abdomen had been opened I do not think it would have been possible to have discovered their existence.

Let us return from this temporary digression to the man whose left kidney was affected with tubercle. His kidney was removed but there was no evidence of any disease affecting the corresponding ureter, that is to say, there was no naked-eye evidence. There may have been, and I think I may say in the light of the subsequent history of the case there probably was, some infection of the walls of the ureter at that time which might have been revealed by the microscope. However, the wound healed rapidly and the patient left the hospital apparently quite well.

For fully three years I lost sight of him. One day I met Mr. Willett in the square and he said to me: "I have an old patient of yours in my wards; I think you would like to see him," and we went up together to look at him. The patient, whom I had believed to be cured, had a large hole in his side occupying exactly the seat of my operation wound. It was four or five inches long and very nearly one and a half inches deep, and was covered with flabby unhealthy-looking granulations. A probe could be passed at least another inch down in the direction of the ureter. The patient was a good deal thinner than he had been when I last saw him, but he complained of little or no pain. There was a hard cord-like swelling to be felt through his abdominal wall, which we took to be a thickened ureter. He was passing no pus with his urine, and had no evidence of any other lesion. He stated that this ulceration began about three or four months previously and had steadily progressed until it reached the condition which I have just described to you. Mr. Willett asked me if I would like to take the case over and I readily agreed to do so. He was under my care in the hospital for nearly two months and then went to a convalescent home at the seaside for a further period. He put on flesh and his general condition improved, but the local trouble remained much the same in spite of scraping and other local treatment.

Off and on he was under my care at this period of his illness for nearly two years, at the end of which time I lost sight of him.

Five years later I was walking home one afternoon from the hospital when a man accosted me and I at once recalled my old patient. He stated he was in perfect health and for the past three or four years had been earning his living by working a sledge-hammer driving steel wedges into the concrete which has to be broken up when pipes are laid along the streets. I need not tell you that this was pretty good evidence of health and strength. I asked him to come and see me at the hospital, which he did a few days later. He has been to see me on several occasions since. The last time I saw him was about six months since. His health is excellent, his side is soundly healed, and he is married and has several children.

I do not think you could find two cases that better illustrate the general course of a local tuberculosis of the kidney when it is subjected to surgical operation.

Now let us turn to another aspect of the disease, and we shall find quite another picture.

Within this last year I had a patient in Sitwell Ward whom some of you, I am sure, will remember, as you felt his prostate with your fingers, and I gave you the reasons which led me to feel sure it was tubercular. He was sent me by an old Bart.'s man, and within the last few days I have heard something of his subsequent history, and I am afraid I must say that his life-history is well-nigh completed.

He is now twenty-seven years of age, and the first evidence of tubercular trouble was a swollen testicle. He had had gonorrhœa a short time previously, so that but little attention was at first paid to his testicle. In June, 1907, the swelling of the epididymis, its painless character, and the total absence of any urethral discharge all pointed to a probable invasion of tubercle. Whether the attack of gonorrhœa had predisposed him to it I will not now discuss, but there is good reason, I am sure, for supposing that this is sometimes the case. A tubercular enlargement so often succeeds an attack of gonorrhœal epididymitis that I am inclined to think these two diseases may act to one another in the relation of cause

and effect. For two or three months more he went on with his work as a carpenter unaware that he was suffering from any serious ailment.

Towards the end of August, 1907, he noticed his right wrist began to swell, and you may remember that when he was in the hospital the synovial membranes of the flexor tendons were swollen in both hands just where they pass under the annular ligament. The right hand, which was the first to show signs of enlargement, was the more advanced of the two, and fluctuation could be obtained between the front of the palm and the forearm just above the annular ligament. The swelling was practically free from pain, but it limited the usefulness of his hand. It was a typical case of compound palmar ganglion.

His right shoulder at the same time showed signs of fixation, and there was wasting of the muscles surrounding the joint. This wasting was especially well marked in the deltoid. There was as well considerable limitation of movement.

These were all evidence of a general tubercular affection, but they were so far as he was concerned quite subsidiary to his main trouble, which was frequency of micturition. This began, as you will remember, early in October, and as I think you will also recollect, it was not till he had been in the hospital some few days that we discovered the shoulder and hand conditions which I have just related to you.

You will see then that within four months of the testicle being affected frequency of micturition supervened, and when we looked into his bladder with the cystoscope the mucous membrane was in places very hyperæmic, but there was no actual ulceration. The prostate was markedly enlarged on the right side. I do not know that I ever felt a more typical instance of a prostate enlarged by tubercle. The patient was thin and his corresponding vesicula seminalis could also be easily touched through the rectum and gave evidences of marked enlargement.

I pointed out to you that local treatment would prove of little if any avail in a patient who was so generally infected with tubercle, but we administered some of Koch's new tuberculin in the hopes of possibly being able to raise his resistant power.

He left the hospital after a few weeks and went into the country, but apparently did not gain much by his stay, and I hear that he is now confined to bed practically unable to hold his water, which runs away from him and is loaded with pus and urates. He has wasted almost to a skeleton, and apparently has not more than a few weeks to live. Curiously enough his synovial affections do not, I understand, appear to have made much progress.

You will note that in this case the urinary tract, I might add the genito-urinary tract, was attacked at its outlet and the mischief is travelling up, reaching the kidneys last of all, not one of them but both almost simultaneously.

In some respects this patient's disease is following much the same course as that of our little humpbacked friend in the first bed in Sitwell. He, you will remember, suffered when quite a child from Pott's disease of the vertebral column, and now at the age of fourteen is dying of tubercular disease of the urinary tract, which began, as in the previous case, with frequency of micturition. This condition has lasted upwards of two years and yet at times he seems for a few days to lose almost all his symptoms.

What, however, is so noticeable in his case, and I believe in almost all cases of a similar nature, is the temporary exacerbation which occurs as more and more of the kidney tissue is invaded. If you look at a typical specimen of a tubercular kidney in a museum you will see that its interior is gradually being hollowed out by the tubercular process which slowly invades it. The *débris* thus formed is at intervals passed down the ureter into the bladder and voided with the urine. As each discharge occurs, laying bare more and more of the kidney secreting substance, a rise of temperature usually occurs, so that one can generally predict with a fair amount of certainty when masses of *débris* containing pus are likely to be passed. The passage of these masses often gives rise to symptoms of renal colic and may be associated with the passage of phosphatic stones, which have been formed as a secondary consequence of the diseased condition which I have just described to you.

And this brings me to another point on which I want to lay emphasis, viz. that an ascending tuberculosis in the urinary tract is far more fatal and runs a totally different course from those cases which I first related to you.

Remember I am only telling you that this is a usual state of affairs. It is by no means universal, and the more you have to deal with patients whose

means enable them to obtain plenty of fresh air and the best of treatment, the more you will realise how amenable some conditions are if the best kinds of treatment are bestowed upon them.

Let me shortly relate to you a case which will explain exactly what I mean. Some years ago I was asked to see a young man whose testicle was undoubtedly affected with tubercle with a view to deciding whether he should part with it or retain it. There was no question that he was a tubercular subject, for early in life he had suffered from enlarged glands in the neck which had suppurated. He had had signs of tubercle at the apex of his right lung, and now at the age of thirty-one the only functional testicle he had got was the seat of tubercular trouble. His other testicle was atrophied and small, scarcely bigger than that of a child three years old, and gave no evidence of testicular sensation. The cause of this atrophy was not clear; it was probably due to a congenital lack of development. At any rate so far as outward appearance went he had only one working testicle, and that was probably tubercular. He was heir to a small estate in the country, and both he and his friends were desirous that he should have a posterity to leave it to. He suffered from a certain amount of frequency of micturition, but there was no enlargement of the prostate to be felt *per rectum*. It was probable, therefore, that his bladder was at this time affected with an early stage of tubercular disease. It was eventually decided that he should keep his testicle, and, as he is now the father of four healthy children, it has proved to be of some use to him. It is interesting to turn aside for a moment to consider what steps were taken to preserve it, and restore him to health. He was directed to lead as far as possible an open-air life. To start with he went several sea voyages, spending practically a year at sea, and he came back in the best of health. His testicle had quieted down, and beyond a few nodular masses in the epididymis there was nothing to distinguish it from an ordinary healthy testicle. He lives as far as possible in the open air; of late years, since the open-air treatment of tuberculosis has come more to the front as a means of treatment, he has more than ever led a life in the open air, and has so large a window in his bedroom fully open at night that he may be said practically to sleep in the open air. He cannot be described at the present time as a very robust man, but he rides, shoots, and generally amuses himself, and at the same time realises that it is only by practically living an open-air life that he can retain his health, such as it is. He is now forty-three, and there is no reason why he should not live to a good old age and die from some disease totally unconnected with tubercle.

There is yet another phase of tubercular disease of the urinary tract which was formerly reckoned amongst the most fatal of these affections, but now is capable at least of temporary relief, possibly of permanent cure.

Some of you have, I am sure, seen a young woman who comes to see me periodically and who now looks in excellent health, though I have often stated to you that she is the subject of urinary tuberculosis. About two years ago she was suddenly seized with pain in the region of both kidneys accompanied by frequency of micturition, and was admitted into Laurence Ward as a case of acute cystitis of unknown origin. A culture was taken of her urine and submitted to pathological examination. It was found to be swarming with tubercle bacilli. I want to draw your special attention to this fact because the diagnosis of urinary tuberculosis can so rarely be made by this means. This abundance of tubercle bacilli in the urine was not an isolated occurrence on the special day when the urine was submitted to examination in the pathological laboratory; it occurred day after day during the greater part of the five or six weeks which she spent in hospital. There was no secondary infection whatever. The urine was infected with a pure culture of tubercle bacilli. You may remember that we administered some of Koch's new tuberculin, $\frac{1}{10000}$ mgrm., every fortnight for some length of time, and under this treatment she speedily improved, and has remained well with sterile urine for more than six months since her injections were discontinued. You will perhaps ask me how long she will remain so? That is a question which I cannot answer. I have, during the last few years, seen several such patients, and one I have under observation who has remained well for more than three years. Before the advent of the new tuberculin I should have said such a case would prove rapidly fatal. I have known similar cases die with symptoms practically of suppression of urine in less than six weeks from the onset of the disease, and a post-mortem on such

a case usually reveals two kidneys stuffed with miliary tubercle and possibly a bladder affected as well, but this new tubercular vaccine works a very markedly beneficial effect on all such cases.

If you reflect on these cases which I have brought before you in my clinical lecture to-day you will, I am sure, realise without difficulty how serious a condition is tuberculosis of the urinary tract, especially when it is the ascending variety which you have to deal with. There is, however, I am sure, no reason for taking too despondent a view of its prognosis if only you attack it in time and deal with it vigorously. Vigorous treatment amounts to prescribing abundance of fresh air, and, what is more, seeing that your patient gets it. It is not sufficient merely to prescribe it vaguely as a sort of pious wish and hope. You must not only lay down the principles but see that the details are carried out. Above all things do not be afraid of your patient catching cold; wrap him up warmly and he will take no harm out of doors in any weather, provided he is protected from the rain. Let his meals be served out of doors, and keep him quiet and restful, and you will be surprised how the worst cases improve and the less severe ones make a good recovery. Let active surgical treatment be undertaken only when it is urgently demanded to drain abscess cavities or remove hopelessly diseased parts. Your difficulties will lie with the patient's friends quite as much or even more than with the sufferer. He will, if he is seriously ill, gladly do what you wish. His friends may accuse you of hastening his end. No cases demand more care, watchfulness and courage on the part of the practitioner, and if you exercise these qualities you will be rewarded by success in a considerable number of cases.

November 9th, 1908.

We have received from Burroughs Wellcome and Co. specimens of "Tabloid" hypodermic, No. 121—atropine sulphate, gr. $\frac{1}{200}$, strychnine sulphate, gr. $\frac{1}{100}$; and "Tabloid" hypodermic, No. 122—atropine sulphate, gr. $\frac{1}{150}$, strychnine sulphate, gr. $\frac{1}{80}$. These are of service in the treatment of inebriety and narcomania. The hypodermic administration of small doses of atropine and strychnine for a few weeks has been found to create a repulsion of alcohol more or less lasting in character. Owing to their precision of dosage and solubility these preparations present a convenient and efficient means of treatment.

COLOSTOMY BY A NEW METHOD, WITH ATTEMPT AT SPHINCTERIC CONTROL.

By CHARLES RYALL, F.R.C.S.,
Surgeon to the Cancer Hospital, Brompton, S.W.

Colostomy as a surgical procedure has been practised for considerably over a century, and yet the great majority of operation cases still suffer from what has always been the main drawback to the operation, and that is the absence of adequate control over the escape of the intestinal contents.

For this reason many patients shrink from submitting themselves to such an operation, and others postpone its execution far beyond the time advisable. Even many of those who have the operation carried out regard themselves, more or less, as outcasts.

Nevertheless, colostomy, as a means of saving and prolonging life, is an exceedingly valuable surgical measure.

Could we but ensure proper sphincteric control and guarantee this to the patient, the operation would be robbed of its chief horror. To this end many varieties of operations have been devised and carried out. In some cases fairly good control has been obtained, but the majority have been sadly lacking in success. Even in those where the best results have been obtained the operations have been severely handicapped by the exceeding complexity of their technique.

Two methods are now generally adopted for dealing with the bowel in colostomy. In one a spur is drawn through the abdominal wall; and in the other the bowel is divided, the lower portion is closed, and only the upper segment of the bowel is used to form the colostomy.

The spur operation, because of its simplicity and rapidity of execution, is by far the most popular method. At the same time it generally gives the least satisfactory results with regard to control over fæcal discharge, and this is due to the great bulk of the spur and, consequently, the difficulty of compressing or controlling such a mass by any muscular arrangement.

On the other hand, and for the reverse reason, better control is generally obtained in those operations where the bowel is divided and where only

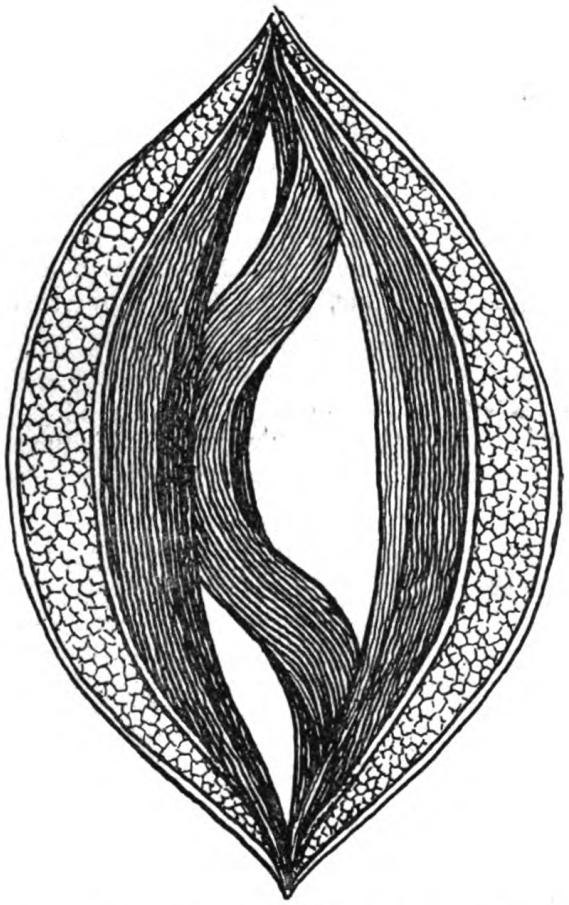
the upper segment is drawn through the abdominal parietes.

Without discussing the various methods which are at present practised, I will now give details of the operation which has been adopted by me, with a view of attempting sphincteric control.

The colostomy is made through the rectus. This is a muscle which is well developed in most subjects, and remains so, in the majority of cases, until advanced emaciation takes place.

Howse first recognised its value, and therefore

FIG. 1.



recommended that colostomy should be made through this muscle, with the hope that muscular contraction would effect a fair amount of control. By his method the bowel was drawn through an opening made by splitting the muscle-fibres vertically.

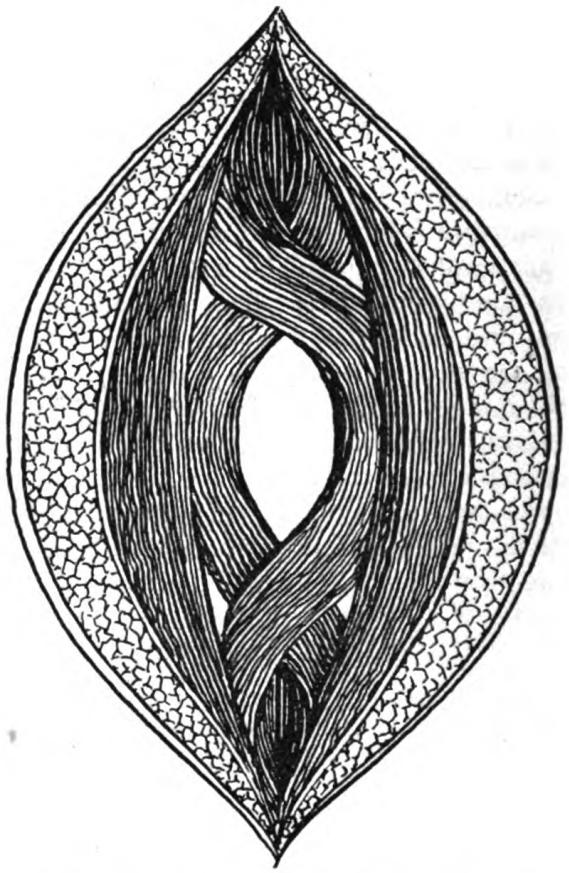
Von Hacker, later, suggested that better control could be obtained by drawing the bowel obliquely through the muscle.

Both these operations provided a certain amount of control, but not to any great extent.

New Method.

An incision three inches or more in length, according to the thickness of the abdominal parietes, is made through the lower half of the left rectus parallel with, and one and a half inches to the left of, the mid-abdominal line. It is advisable to go through this half of the muscle, which consists of long strands of muscle-fibre, whereas the upper portion is broken up by linea transversa. The anterior part of the sheath of the rectus is divided the whole length of the wound, and the

FIG. 2.



fibres of the muscle are split longitudinally for a distance of about two and a half inches. The fascia, transversalis, peritoneum, etc., are then incised, and an opening is made sufficiently large to admit of digital exploration of the abdomen.

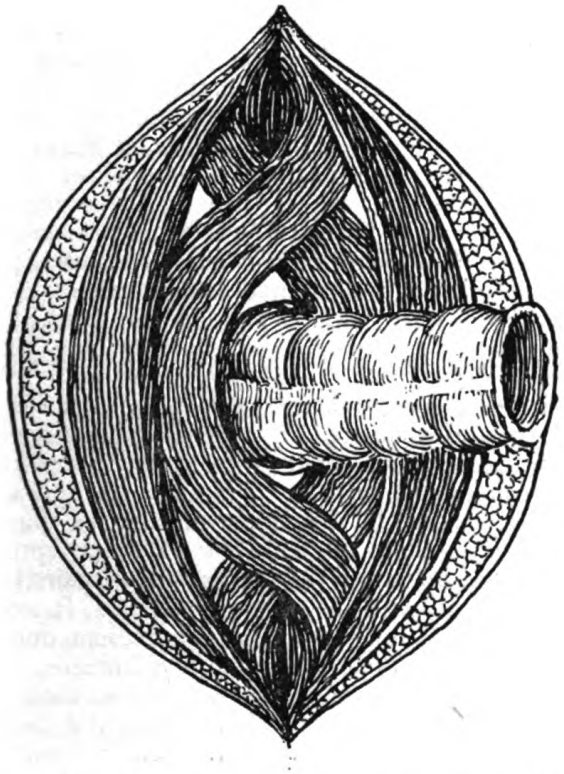
Treatment of the bowel.—The pelvic colon, or that part of the large intestine which is selected for colostomy, is sought for and drawn out of the wound.

After taking the usual precautions to protect the wound against fæcal contamination, the bowel is

divided across at such a point as to permit of the upper segment being fixed in position without any undue tension. The lower part of the bowel is then closed by suturing; and this is readily effected by first of all inserting a continuous suture, and then invaginating the cut end of the lower segment into the bowel by means of a modified purse-string suture.

It is important that when the upper segment of the bowel is fixed in position it should be free from all tension. Should it prove to be tightly bound down by a mesentery that is inconveniently

FIG. 3.



short, this difficulty can be overcome by dividing the latter to a sufficient extent.

The gut is then rendered less bulky by ligaturing and removing the appendices epiploicæ, and also as much mesenteric fat as possible, but in carrying this out, and likewise in freeing the mesentery, it is essential to guard against any serious interference with the blood-supply. The less bulky the gut, the more readily can it be controlled by the new sphincter.

Formation of the Sphincter.

This is constructed from the fibres of the rectus by a method of myoplasty.

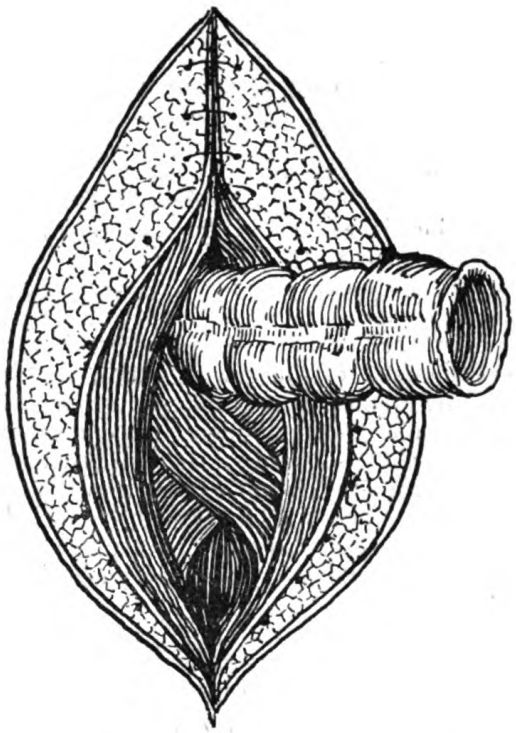
Where the rectus has already been split a stout loop of fibres is detached from the posterior part of the muscle, on each side of the line of cleavage (Figs. 1 and 2).

Each loop is then drawn over to the opposite side, and made to overlap. By this means these two overlapping loops form a ring of muscle-fibres, and through this the gut is drawn (Fig. 3).

Sutures are then inserted so as to bring together the separated muscle-fibres above and below the gut (Fig. 4).

The anterior and posterior fibres of the rectus at each end of the wound are brought together

FIG. 4.



with a separate suture, and when these are made secure the attempt at a new sphincter is completed.

It will thus be seen that the sphincter consists of two lots of fibres, viz. longitudinal fibres, provided by the anterior part of the rectus, and encircling fibres, formed by muscle-loops of the posterior part of the rectus.

The bowel is then anchored in position by two sutures passing through its sero-muscular coat, and also through the abdominal parietes.

I also make it a practice, after having sutured the skin, to fix the gut to the latter with a few sutures.

During the process of healing a certain amount of retraction of the gut takes place, and therefore, in performing the operation, it is essential to leave a certain amount of gut protruding; in fact, it is better to err on the safe side and leave too much, as any superabundant portion can be readily removed when the process of healing is complete.

In dealing with the muscle-fibres, either in splitting the rectus, or in detaching the loops, the greatest gentleness is called for. Any undue roughness or unnecessary separation of muscle-fibres may cause serious interference with their nerve and blood supply, leading to permanent damage and consequent atrophy of the fibres.

Of course it is a debatable question as to whether atrophy of the muscle loops eventually takes place. Probably it does in some cases, where, owing to injudicious separation or damage to the fibres, their nerve and blood supply has been seriously interfered with.

Although it is early to speak, yet I am of the opinion that atrophy will not occur if due care be taken.

In my experience up to date the amount of control obtained by this method is encouraging, but whether it will stand the test of time still remains to be proved.

I regard it of considerable help to the patient to describe to him what has been done, and explain that muscular contraction is the means of maintaining control.

It is my opinion that the best results in these operations are obtained where it is carried out in connection with the rectus, but a similar measure can be adopted wherever gut is drawn through muscle.

The same method of obtaining control might also be tried in cases of gastrostomy and appendicostomy.

November 9th, 1908.

We have received from Cassell and Company a copy of 'Estimation of the Renal Function in Urinary Surgery,' by J. W. Thomson Walker, M.B., C.M.(Edin.), F.R.C.S. The book is a valuable and interesting clinical study of the present means of estimating the renal function in surgical diseases of the urinary organs, which involves a study of symptomatology and of the application of physiological methods to diagnosis in urinary surgery. Mr. Walker has most certainly succeeded in producing a volume of great utility in practical work. The lucid manner in which he brings out his points is refreshing. It is interesting to note from his work that he examined 109 cases of simple enlargement of the prostate with a view to ascertaining whether symptoms of renal failure were present. In all these cases the obstruction was sufficient to interfere with the complete emptying of the bladder. The amount of residual urine varied from a few ounces to complete retention. Some of the patients were unaware of the residuum; others had their bladder greatly distended, and the urine dribbled away in passive overflow. Others, again, passed a catheter occasionally, and some were absolutely dependent upon this means of voiding urine. Thus the amount of obstruction, if measured by the condition of the bladder, varied, and the pressure against which the urine was secreted might be expected to vary in similar measure. The symptoms of renal inadequacy did not, however, always follow the variations in the mechanical defect in the bladder. They might be well marked with an ounce or two of residual urine, and poorly developed or quite absent when the bladder was fully distended. Thus no symptoms of renal inadequacy were present in twenty-five of these cases (22·9 per cent.), and the bladder was fully distended in three of these, and the patient dependent upon the catheter for relief in four. In 100 cases of stricture of the urethra attending his out-patient department at St. Peter's Hospital, sixty-five had no symptoms of renal inadequacy and thirty-five had more or less marked signs. Some of those with symptoms of impaired renal function were narrow strictures, admitting only about a No. 6 French bougie (nine cases). But, on the other hand, symptoms were marked in some patients with stricture admitting large steel bougies (13–15 and 14–16). Pain was one of the most frequent signs of renal disease in these cases. The pain was situated, in the greatest number of these cases, at the angle formed by the erector spinæ mass of muscles with the last rib, and was usually localised by the patient tucking his thumb into this spot. Occasionally it was diffusely spread across the loins in this region. The pain had usually a constant, dull, aching character. In a few cases of stricture the pain was only present during micturition. In one patient, who volunteered this information, the pain gradually diminished during intermittent dilatation of his stricture, and finally disappeared completely. Mr. Walker regarded this type of pain as an indication that the sphincter action of the lower end of the ureter had been destroyed by the back pressure of the urine. The most frequent symptom of interference with the renal function was thirst. A dry tongue is an important index of the state of the renal function. Headache is a feature of many of these cases. In the later stages of "urinary septicæmia" the face has a peculiar yellow, earthy look that is almost characteristic. Associated especially with septic renal changes, hiccough is a sign of grave import. Its incidence after an operation upon the lower urinary tract may be looked upon as a signal for alarm. In the chronic cases of aseptic or septic pyelo-nephritis there is nothing to warn the surgeon of the delirium that may sometimes follow surgical interference, even of a minor degree.

THE CLINICAL JOURNAL,

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A CASE OF ANKYLOSTOMIASIS.

A Clinical Lecture delivered in the London School of Clinical Medicine.

By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Consulting Physician to St. Bartholomew's Hospital; Senior Physician to the Seamen's Hospital, Greenwich.

GENTLEMEN,—I propose to discuss with you to-day the case of A. J—, æt. 46 years, a fireman, who was admitted here under my care some time ago. He complained of increasing general weakness and pallor, pains in the head and giddiness, especially on stooping, the pain being of a shooting character on the left side and in his left eye; further, of indigestion and regurgitation of part of his meals half-an-hour afterwards. His legs swelled at night. He had suffered from enteric fever twelve years ago, and from ague seventeen years ago. No history of syphilis. Family history satisfactory.

The general nutrition was not good. The muscles were in poor condition, but there was plenty of superjacent fat. Pallor of face, buccal membrane, and nail-beds. The heart-sounds were pure but feeble. Nothing of importance was to be noted in the lungs. There was some tenderness over the right lobe of the liver, and a suspicion of a lump in this situation on full inspiration. Tenderness also on pressure at the epigastrium. No enlargement of the stomach. Spleen not palpable. The urine was natural. There was slight general improvement after admission, but the tenderness of the abdomen remained, and was felt also in the right loin and iliac region. The bowels were constipated. A test meal showed a trace of hydrochloric acid (·08 equivalent), no sarcinæ, and a few torulæ. The symptoms of progressive weakness and anæmia, together with the hepatic pain and dyspepsia, suggested the possibility of abdo-

minal malignant disease. Careful dieting and some iron and arsenic were prescribed. An examination of the blood made shortly after admission by Mr. L. Nicholls in the Pathological Laboratory of the hospital showed red globules 3,500,000 per c.mm., hæmoglobin 80 per cent., with poikilocytosis and variations in size, but no nucleated red globules; while for the leucocytes there were polymorphonuclears 64 per cent., lymphocytes 28 per cent., hyaline 3 per cent., and eosinophiles 5 per cent. The increase in the latter suggested to Mr. Nicholls the likelihood of ankylostomiasis, and this led to an examination of the fæces for ova and other evidence of this parasitic invasion. Inquiry showed that this man had been engaged in some railway work in Mexico, and had had a rough time there, often having to drink river waters of an unsatisfactory character. The first, and many subsequent, examinations revealed the presence in large numbers of the ova of ankylostomes, and there were also numerous specimens of the little worm discharged. Ova of *Strongylus subtilis* and of a trichocephalus were also found.

We had thus sufficient evidence of an abiding cause for the anæmia and most of this man's symptoms, and forthwith began treatment for removal of it by means of dosage with thymol. After an aperient, and with restricted liquid diet, which is imperative, thymol was given in doses of 15 gr., later of 30 gr., followed by castor oil.

The effect was marked, and repeated examinations of the fæces showed a decided diminution in the number of dead worms and ova discharged within a week.

This thymol treatment gave rise to no disturbance, and was well borne. After three weeks Betanaphthol was substituted for it in doses of 15 gr., followed by castor oil, because only a few ova were then being discharged. More came away as the result of this treatment. The patient took daily a mixture containing iron and arsenic in compound decoction of aloes, and had a good diet.

There are still ova and dead worms to be found in the fæces. The patient's general condition has improved, but he is still anæmic. There is a slight systolic murmur at the apex of the heart, and a loud venous hum is heard in the jugular veins. No souffle at the base of the heart, and no dilatation of its walls. The pulse is of low tension. Pupils rather large. The abdominal pains are no longer felt. A blood-count on September 23rd showed reds 4,000,000 per c.mm., polymorphonuclears 50 per cent., lymphocytes 32 per cent., hyaline 3 per cent., and eosinophiles 50 per cent.

This particular parasitic invasion has been recognised for seventy years. It is widely spread east and west in tropical and sub-tropical countries, and cases of ankylostomiasis may in these days of rapid locomotion be met with anywhere, although the disease is not endemic in this country, so far as is known. Its discovery in the workers of the St. Gothard Tunnel in 1880 led to an awakening of interest in the subject, and the later detection of it in 1902 amongst some Cornish tin-miners brought the matter home to us more closely. The disease is most prevalent in Egypt, and is known as Egyptian chlorosis. The parasite is a small nematode thread-worm, the male 6 to 11 mm. long, and the female 7 to 15 mm. in length, and the latter are thrice as numerous as the former. They are white when seen alive, grey when dead, and brown when full of blood. The posterior end is the largest, the neck narrow with a bulging capsule, armed with four hooks and two teeth. The female produces innumerable eggs. The habitat is chiefly the jejunum, and although the worm is termed *Ankylostomum duodenale* it is less frequently found there and rarely in the ileum. It clings to the mucous membrane by its hooks and teeth and sucks blood freely. The numerous eggs are discharged with the fæces and readily found by the microscope. They are oval in form, with a transparent shell, and contain a segmented yolk of from four to sixteen cells. These eggs develop no further within the body, but rapidly pass on to a larval state, living in mud or water till they re-enter the human host.

The symptoms of the disease induced are in relation to the amount of invasion by the parasite. If very numerous, the amount of blood abstracted in the bowel becomes serious and leads to grave anæmia. In the case of a small invasion in healthy subjects the anæmia may be slight and unnoticed, and so many cases of this disease pass without recognition or suspicion in countries where the disease is prevalent. The cachexia and anæmia which result in grave cases are sometimes attributed to malarial influence. The absence of ague attacks and of an enlarged spleen, with a careful examination of the blood, afford plain evidence against this view. The anæmia may lead to fatty and dilated heart and similar degeneration of the liver and kidneys. There is no associated pyrexia.

The abdominal pains and dyspepsia which led this man to seek advice are recognised amongst the symptoms of ankylostomiasis. They are readily explained by consideration of the condition of the small intestines in such cases. The continuous numerous punctures and resulting ecchymosis of the mucous tract may well give rise to discomfort and pain. It is easy to understand how such symptoms may be variously attributed to any but the right provocative, and the true nature of the case be ignored. The lesson for us here is to bear in mind the possibility of this invasion in patients suffering from tropical anæmia, and both by examination of the stools and investigation of the leucocytic blood-count to recognise or exclude the existence of ankylostomiasis.

The detection of excess of eosinophile-cells in the blood of patients thus affected is important and suggestive. It plainly put us on the right track for diagnosis in this case. These cells may increase to the extent of 30 to 50 per cent. of all leucocytes in some cases. The cause of their production in such numbers is not known, but they are apt to be excessive in other morbid states, and have been thus found in the subjects of asthma, in filariasis, Bilharzia, and trichiniasis, also in some skin diseases.

Thymol is probably the best remedy. Santonin, oil of male fern, and beta-naphthol are also of use. Treatment must be assiduously carried on to promote recovery. The anæmia demands the employment of iron and arsenic and a good diet, including some wine. It is apt to be tedious.

Prevention however, here, as always, is better than cure. This signifies attention to water-supply, especially in tropical and sub-tropical regions.

If the inhabitants of such countries habitually boiled their water and their milk a large number of the ailments prevalent in those latitudes would be unknown. The fæces should be powerfully disinfected with carbolic acid or iron-sulphate, or be burned with petroleum and sawdust. They should never be buried or discharged into drains.

Take note of the fact that this man is not yet cured of his malady. He is anxious to leave the hospital, but he requires continued treatment at intervals with thymol dosage, and I fear that if he goes out he will remain a victim of the disease. I have a rooted objection to partial cures of any ailments that are amenable to treatment, however prolonged it may be. Our object is always to promote sound recovery, and not mere alleviation, wherever that is possible.

I show you here specimens of the worms and ova and also one of the blood, illustrating the degree of morbid eosinophilia. Mr. Lucius Nicholls has kindly furnished these to illustrate my lecture to-day.

November 16th, 1908.

EMPLOYERS' LIABILITY FOR INJURY OR ILLNESS.*

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

SINCE the passing of the Workmen's Compensation Act in July, 1907, particular interest has been attached to the question of liability of employers in cases of injury or illness sustained or contracted as a result of certain employments, and there is no more competent authority on this subject than Sir Thomas Oliver, of Newcastle-on-Tyne, who has, on many occasions, been appointed by the Government as an expert to sit on Commissions to investigate the evils attendant on certain employments.

It is from his reports and his very valuable work, 'Diseases of Occupation,' that I have gathered most of the information I am going to speak to you about, and which will, I think, interest you.

The annual report of the Chief Inspector of Factories and Workshops for the year 1907 tells us that: "While recent legislation has tended to impose additional duties and responsibilities upon certifying surgeons, it has also led to an increase in the total number of accidents and cases of trade poisoning reported."

By drawing attention to the efforts made by the employers of labour to mitigate the dangers of certain industries, by showing how loyally they have tried to co-operate with the Government in following the prescribed regulations, and in giving information which has led to the enforcement of such regulations, often at the expense of their own pockets, I hope that a heartier co-operation and a greater amount of mutual trust will grow up between employer and employed, for this is an essential to ensure the smooth working and success of all regulations.

I am speaking, of course, of the good employers, for there are still men and women who try to evade the laws made for the benefit of all, and if the Workmen's Compensation Act hits them hard it will be a matter of small regret to any of us.

I have said that there must be mutual trust between employer and employed. There is another essential. The workman or workwoman must

* A lecture delivered at Gresham College.

have the necessary education and intelligence to understand the dangers of his or her employment, and must follow certain hygienic rules necessary for everyone to keep in good health. No amount of parliamentary legislation will help a man to live a healthy life who neglects personal cleanliness, who does not obey the rules laid down for his welfare in the factory, who sleeps in an atmosphere saturated with the poisons emanating from his own body or from dirty clothes, who tries to keep himself warm by shutting out the health-giving air which should enrich his blood, who drinks to excess, or who eats bad or insufficient food.

Accidents occur most often when the workman is tired, towards the end of the day or when his health is rendered feeble by some illness, for fatigue leads to carelessness, and we can well imagine how a momentary forgetfulness may have disastrous results. Fatigue, again, makes the workman less resistant to bad weather, and overworked groups of muscles are more liable to paralysis than those used more reasonably.

I need hardly insist on the fact, which you must all realise, that bad hygienic habits are a predisposing factor, if not a cause, of many diseases contracted during employment. There is nothing that induces to certain forms of industrial poisoning more than alcoholism, besides being a frequent cause of accident. An intemperate man should never be accepted in any employment where there is the slightest danger of lead-poisoning, such as lead-mining, smelting, the manufacture of pottery and earthenware, enamelling, dye works, painting, and many other trades, for he will be the first to fall a victim to that dread disease. Also the excessive use of alcohol leads to carelessness and recklessness, causing a man to run many a useless risk. Again, it is the cause of sudden attacks of giddiness or faintness, which may be the explanation of many terrible and unaccountable falls, not following a fit of drunkenness, but being the result of a system slowly poisoned by continual indulgence in alcohol. The employer can only guard himself against such liability to accident by care in eliminating those workmen who are intemperate in their habits.

In spite of the utmost precautions in the fencing of machinery, and rules to ensure the safety of those employed, the reported accidents still keep increasing, which is partly attributed to the greater amount of machinery. But there must be increasing wear and tear to the nervous system of factory workers from the growing speed of machinery, and there is no doubt that the nervous strain tells very heavily upon them. In the case of women, this affects, not only themselves, but their future offspring, and it is a very serious question for the race. To quote Sir T. Oliver: "Will this speeding up tend to make female mill workers better mothers, and help them to give birth to healthy and robust children, or to infants who are puny, ill-nourished, and of a highly-strung nervous system? In some American factories in which stitched muslin underwear is made, so great has been the improvement in the machinery of late that the sewing machines are carrying from two to ten needles instead of one as formerly, and as a consequence many of the girls are no longer capable of the sustained effort necessary to follow the increased speed, and have been obliged to relinquish their occupation; the strain of the eyes in watching for broken threads in order to stop the machinery is almost intolerable; it requires an amount of nervous energy and constancy of attention which the operators cannot supply. There is a limit beyond which the speed of machinery cannot be run without detriment to the health of the operators, unless their hours of work are materially shortened." *

Hurry is a growing evil, and each generation makes greater demands upon its nervous energies. We get through more in the day than did our forefathers, but are we paying too high a price for it in fatigue and early exhaustion? The quicker a machine can work the cheaper can the article it produces be sold, but there is a human sacrifice which is not sufficiently considered. If the nervous strain is great, fatigue is greater, and the body and mind require more rest, and thus we find some factory workers spending half their Sundays in bed. This is a wise course to pursue, but just as often do we find these over-wrought workers seeking recreation and change, instead of rest, at music-halls and other ill-ventilated places of entertainment, and this must be condemned as utterly unwise; temporarily it may seem a rest, but it can be of no real benefit to the tired system.

Nature does her best to accommodate herself to the special needs of the individual worker, and by gradual training we may bring our muscles, our

* 'Diseases of Occupation,' p. 3.

heart, and even our nervous system to withstand the strain thrown upon them, whilst another worker, untrained, would break down under the same conditions. But often what causes the labourer to succumb is not the hard work, but alcoholic excess, fatigue produced by the insanitary conditions of his home, or some unhygienic folly which causes an unnecessary strain to be thrown on his organs, which would otherwise bear up well under his work.

I know that it is easy to speak of the necessity of healthy homes, to condemn the overcrowding of sleeping-rooms, and to insist upon the importance of good food, but it is very difficult, if not impossible, for the poorer artisan always to obtain these. For this reason it is all the more important that factory laws should be made to insist upon healthy conditions in factories and workshops. If it is necessary or advisable in certain employments, which I will refer to later on, for the workpeople to wash, to change their clothes, or to bathe after the day's work, proper accommodation, hot and cold water and soap must be provided; there should be supervision to insist upon all precautions being observed; in fact, it should be made easy and not difficult for the workers to follow the rules laid down for them.

The dangers in all factories that the employer should guard against are imperfect ventilation, absence of means to withdraw dust, and insufficient washing appliances.

We will now consider the question of disease arising in the wake of accidents, and possibly a result of those accidents. The question of cause and effect has become more urgent in these cases since the passing of the Workmen's Compensation Act, and an answer must sometimes be given when it is exceedingly difficult to decide whether the one has any connection with the other.

For instance, here is a case which was sent to me for an opinion quite lately, exactly two years after the accident.

The man, who was 58 years old at the time of the injury, which caused several of his ribs to be broken, had been under medical treatment for all the two years, and had been drawing a weekly pay from a certain large company. He seemed to me to exaggerate all his symptoms, and I found that in addition to the pain in the side of which he complained he had disease of his arteries and kidneys, while there was some delicacy of his heart and one lung. It was obvious that the lung condition might be due to his broken ribs, which had, of course, long ago been cured, but I believed that his other diseases were due to premature old age and not to the accident. The question in my mind was what view a magistrate or a jury would take of the case, and when pressed for my advice I recommended a compromise. I have no doubt the man got liberal terms from the company in consequence of my stating that there were unanswerable arguments on both sides.

I have seen several similar cases, but I will not weary you with them now.

This question has received more attention in Germany, where compensation for injury has been carried further than here.

There is reasonable foundation for a claim for compensation if injury received while at work is the direct cause of tuberculosis or consumption. A severe blow on the chest may be followed by spitting of blood and other signs of tubercle in the lung, and the patient dies some months after the injury. At first sight the answer seems easy enough, but the physician who has to decide the question as to whether compensation is due must be able to know for certain that the patient was perfectly sound before the injury, and this is often an impossible question to answer.

A man may show *no* sign of consumption and yet have latent tubercle in his lung or other organ, which the injury appears to light up, but which, without that injury, might have developed later on. In Germany, ever since 1884 a workman has been compensated for accidents received at work, and the question of "white joint" has given rise to much discussion. A man, for instance, receives a blow or a strain to a joint and shortly afterwards develops white swelling or white joint, which is tuberculous. Now when this develops the patient naturally tries to recall some injury to account for his illness, but it is doubtful whether the injury always *is* the cause of it. Sometimes the employment obviously seems to give rise to certain tuberculous diseases, and, as an instance, we find Perroud, in 1875, "drew attention to the frequency of phthisis among the sailors on the Rhone. These men are of good physique, and they follow a healthy occupation in the open air. Their disease was attributed to the men leaning with

their chest-wall upon the pole of the rudder in steering their ships. It was thought that the repeated pressure favoured the development of lung disease, the interesting point being that the maximum focus of the disease, as revealed by the stethoscope, always appeared to be located at the level of the point of pressure by the pole, viz. under the right clavicle. The sailors themselves were of the opinion that their illness was the result of leaning on the pole."* Here the cause and effect seem obvious, but when the connection is doubtful, where the injury merely seems to have lighted up and intensified a tuberculous tendency, causing active disease, partial compensation may reasonably be claimed.

In Germany the question of compensation is a less personal one between employer and employed, because, as I explained to you in my lectures on tuberculosis last year, the State manages the sick, accident, or old age fund, which simplifies matters in many ways.

Injuries to the abdomen and back cause other diseases, but there, again, an injury may only light up some latent trouble. The Workmen's Compensation Act will give rise to many questions as to whether the injury was, or was not, the cause of many a disease which hitherto has in no way been considered as resulting from injury. Here is a hypothetical case which has been instanced by Sir T. Oliver and others: A man who is known to indulge too freely in alcohol receives an injury. The injury of itself is so slight that under ordinary healthy circumstances recovery would be rapid and complete, but owing to the man's habits delirium tremens develops, and in the course of this the patient dies. But for the accident there would have been no delirium tremens. To that extent, therefore, the injury has determined the malady, but had there been no excessive indulgence in alcohol there would have been no delirium tremens. What will British lawyers make of such an association of circumstances? The French Act of 1898, recognising that the injury had played even a small part in the development of symptoms, would have indemnified the relatives of the drunkard.

Cancer, or malignant disease, often appears to have direct relationship with a blow, but the disease may have existed unsuspected for some time previous to the accident, and only extends to the bruised part as a result of the injury, thereby, perhaps, hastening the end. It must be insisted upon that there should be a post-mortem examination in all cases where the compensation is claimed after death from cancer, which is said to have been caused by injury.

I may refer here to the difficulty the medical man often encounters in differentiating between the true sufferer and the man or woman who is simulating disease, either from want of control, hysteria, or merely from a desire to claim compensation. After a severe shock there may be such a nervous condition, such anxiety as to the future, as to cause the patient involuntarily to exaggerate his symptoms, but the real malingerer is he who resorts to every falsehood and ingenious device to simulate the ill-health he professes, and it is often exceedingly difficult to decide how much is real and how much is false.

People eager to claim compensation after an accident will simulate a loss of power in a limb, for instance, and simulate so well that only under an anæsthetic like chloroform can it be discovered whether the injury is real or not. Blood-spitting is frequently simulated, and when suspicion is aroused and the mouth and throat are examined, the physician will find a piece of raw liver is being chewed to produce the blood, or the roof of the mouth of the patient is found to be bleeding from self-inflicted wounds.

When compensation can be claimed for a disease of the skin, malingering is often resorted to. By mechanical irritation or the use of some chemical certain inflammatory conditions or rashes can be produced, and the physician's suspicions are aroused by the inability of the patient to give a clear history of the condition, or by the general mental state of the patient. The determined malingerer may try almost every means to produce the eruption he wishes to suffer from, and we have cases of injury self-inflicted by pins and needles, by burning matches, or we find black lead or other matter used to discolour the skin.

When the condition of the patient is made the subject of a process in a court of law, the legal adviser for the plaintiff makes it his business to exaggerate every symptom, while those for the defendant do their utmost to minimise the actual disabilities which the plaintiff has incurred. In this way a jury, not possessing any expert know-

* 'Diseases of Occupation,' p. 15.

ledge, is too often made to judge between such conflicting accounts, with most unsatisfactory, and often unjust, results.

The following remarkable case of malingering was published* by Dr. Lachlan Grant and Dr. T. Hay Campbell: The patient, a female, æt. 36 years, had been under constant medical treatment for the past twenty-one years. The last eighteen of these she had spent in bed, and it was extremely probable that she would remain in it for the rest of her life. She was undoubtedly the subject of an inherited neurosis, her mother having suffered from hypochondriasis, and several of her relatives being of a nervous disposition. It was not till she was eighteen years of age that her condition became really troublesome. At this time she took to bed with a unilateral megrim-like headache. From then till now no power of persuasion, no remonstrances—quiet or stern—from medical men, no particular line of treatment, had been able to induce her to leave it. And as the necessity for remaining in bed had to be supported by some evidences of suffering, she resorted to subterfuges, remarkable alike for their number and variety. The megrim-like headaches became more violent. She clamoured for sedatives to relieve the pain, and in spite of her assertion that none of the drugs given could give her relief, she soon manifested a liking for bromide of potassium, which had been occasionally exhibited. Indeed, the consumption of drugs early became a passion with her, and few visits could be made without a "placebo" being prescribed. When the novelty of the headache began to wear off and sympathies became more guarded, an extensive pustular eruption made its appearance on the left leg. This had evidently been brought on by scratching. Boracic and starch poultices, mild antiseptics, and general tonic treatment were adopted, but the condition remained active. Ointments had to be given for years. Again, time outwore the patience of all concerned and a new disease had to be discovered. This cropped up suddenly in the semblance of gastro-intestinal catarrh, epigastric pain, ineffective vomiting. The foregoing symptoms disappeared as abruptly as they had come, and were succeeded by conjunctivitis. Though mild at first, this became, by dint of rubbing, very acute. Bleeding from the gums was brought about by picking them. They swelled, became spongy, and some of the teeth were loosened. All local and general treatment proved useless. In the course of half a year this gave place to a simulation of asthma, with rapid, shallow breathing and a persistent sham cough. Nothwithstanding assurances to the patient and friends that there was nothing seriously wrong, it was evident that to them the terms "medical treatment" and "drug treatment" were synonymous. Consequently many medicines, frequently changed and in homœopathic doses, were administered. Owing to the small importance attached to the pseudo-asthma the urinary system began to rebel. Great pain in the lumbar region, hæmaturia, and passage of stones were the prominent features. These stones were most interesting. For curiosity a few of them were sent to Mr. C. T. Clough, of the Government Geological Survey, who kindly reported as follows: "I have no doubt that all, or nearly all of the small pebbles are composed of quartzite, one of the commonest rocks in your district. There are two, very small, of the nature of which I was uncertain, but they may also be quartzite. If not that, they must probably be classed as natural rocks." She passed these stones at the rate of twenty to thirty per week. Probably she derived her supplies from the soles of her brother's working boots. The sweat glands now took up their *rôle*. Sweating became a troublesome symptom. It resisted all attempts at treatment, but that did not worry her medical attendants. They knew their patient was of a philosophical turn of mind as regarded her bodily infirmities. Uncured ailments were soon forgotten and new ones discovered. Palpitation, shortness of breath, and sinking weakness in the cardiac region made excellent successors to the "drenching sweats." These symptoms quickly subsided. Pustular eczema over the abdomen supervened. This was followed by a troublesome onychia which lasted for six weeks, then joined its predecessors in oblivion. At the present time pains in the joints of both hands formed her chief complaint. This had occupied her attention for the last six months, but a sleeplessness which was becoming more and more persistent was asserting itself and threatening to replace the joint affection. To attempt to give a detailed account of her numerous stigmata was unnecessary. Already enough had been said to

* 'Medical Press,' October 7th, 1908.

show the sad condition to which this patient had been reduced by a neurotic temperament and the free exercise of a morbid imagination. The absolute impossibility of many of her symptoms, and the shamelessness with which she advocated them would be amusing if they were not evidences of a serious defect. For what was the result of it all? A woman who might have led a useful life had been simply a trial and an encumbrance to her relatives. These latter by emphasising her every symptom, promoted a vicious circle, likely to be broken only at her death. Suggestions regarding exercise in a bath-chair or removal to hospital were met by blank refusal. Electro-therapy, psycho-therapy, and treatment by drugs had alike proved powerless to accomplish anything. There was no hope of improvement, and every probability of the patient living for twenty years still. It would thus be seen how serious such a case must be. Drs. Grant and Campbell concluded their report with the following remark: "We have seen many instances of malingering in neurasthenia and hysteria, but we have never happened on one in which the malingering element was so predominant and constant as in this case."

We now come to specify certain occupations with their attendant dangers, the precautions that should be taken, and the results of the neglect of such precautions.

Let us consider the manufacture of phosphorus or lucifer matches. Until recently all strike-anywhere matches were made of white or yellow phosphorus, which is highly inflammable and injurious to health. The red phosphorus, discovered in 1845, is comparatively harmless, and the Swedish, or safety matches, are made from this. They only strike upon a specially prepared surface, this surface containing the red phosphorus, and not the match head, which has a compound rich in oxygen, required to induce the flame. But until recently a great deal more white phosphorus was used in Great Britain than the red.

Our match industry gives employment to more than 4000 people, three quarters of whom are women, and the death of a lucifer match-maker about eleven years ago, and the report that there was much ill-health among the workers, caused the question of their employment to be raised in Parliament, and Sir T. Oliver and Professor Thorpe were appointed by the Home Secretary to investigate the matter. They found that, owing to the fact that the manufacture at first was very simple, and required hardly any machinery, any building was considered good enough, and it was often clandestinely carried on as a domestic industry, to the danger of the workers.

In the mixing of the paste, which contains 5 per cent. of phosphorus, if done by hand and in open vessels, and in the dipping and boxing of matches, the workers used to be exposed to the dangerous fumes.

The disease dreaded by workers in phosphorus is phosphorus necrosis, or "phossy jaw," a very painful affection confined to this trade. It is an inflammation of the jaw bone, continuing for months, till a piece of dead bone is thrown off, either naturally or removed by operation, often leaving the face much deformed. The common entrance of the disease is through decayed teeth, and all the workers' teeth should be kept in good order. When the upper jaw is affected it is sometimes followed by septic inflammation of the brain, which is always fatal, and of fifty-one ordinary cases reported by Dr. Garman nine ended in death. One hundred and five cases of phossy jaw were reported in the latter part of the last century.

There is also a curious tendency in lucifer match-makers to fracture of the long bones. Thus a man will break his thigh-bone in the simple action of raising his leg on to the pavement.

The questions to be decided by the Committee were whether anything could be done to make the manufacture of lucifer matches from white phosphorus more healthy, and whether a substitute could be found for this dangerous substance. The precautions recommended were: That no worker suffering from carious teeth should be admitted to the factory; that there should be a periodical inspection of the teeth; that there should be improved ventilation and less phosphorus used in the paste. Since these recommendations have been adopted there has been considerable improvement, only twelve cases having been reported since 1900, three of whom had been previously affected.

Besides these precautions, the Diamond Match Works in Liverpool have found great improvement since the introduction of machinery for cutting, dipping, drying, and boxing the matches, without their being handled at all.

In France the manufacture of matches is a State monopoly, and workers are compensated for injury to health in consequence of occupation, but the claims for compensation rose to such a height that the Government was obliged to try and improve the industry. In one year the State paid £16,000 compensation to 669 workers in one factory alone. Meanwhile the Belgian government had offered a prize of £2000 to anyone inventing a safety strike-anywhere match free from white phosphorus. France solved this problem by the production of sesquisulphide of phosphorus, and an improvement took place at once. There were no more claims for damages, and where it is used match-making can no longer be considered a dangerous trade. There has been no case of phosphorus poison, explosion, or fire in French match factories since its introduction. Messrs. Bryant and May report also that there is now no sickness among their people, nor can there be. It has caused no great outlay, but by care and scientific invention a complete change has taken place in this industry. The following extract from the report of the Chief Inspector of Factories for 1907 is wholly encouraging: "No case of phosphorus necrosis has been reported in common with lucifer match manufacture since 1904, with the exception of one, in a single factory. The Home Secretary at the end of July introduced into the House of Commons a bill 'to prohibit the manufacture, sale, and importation of matches made with white phosphorus and for other purposes connected therewith.' The bill has been read a first time. It is an interesting procedure, viewed in the light of the report furnished by Professors Thorpe and T. Oliver a few years ago, and marks a new phase in industrial legislation."

Dangerous Gases.

The Home Office regulations compel the owner of a factory or workshop to provide for each person 250 cubic feet of air during the day's work, and as much as 400 cubic feet during overtime, that is, after a ten hours' day. The atmosphere we breathe consists of oxygen, nitrogen, and carbonic acid gas, and the human lungs are constantly adding to the carbonic acid gas in the air, besides which artificial lights also contribute towards it. You all know, of course, that the carbonic acid gas in the air is poison, and that the economy of man or animal cannot stand more than a certain percentage of it in the air without headache, sleepiness, buzzing in the ears, and, finally, loss of consciousness and asphyxiation. It is therefore necessary for the employer to provide for good ventilation in his rooms, not only because he is thereby obeying the regulations, but because in so doing he is keeping his work-people in a healthier bodily condition, when they are less liable to disease and have more energy for good work.

Carbonic acid gas is the commonest of all poisonous gases, and can be an evil for us all if we choose to live in ill-ventilated rooms, but there are other gases more sudden in their results which some workers have to contend with.

Carbon monoxide is described as one of the products of the incomplete combustion of carburetted gas, also of coal and explosives, and you find it in coal mines where blasting has been done. It forms 7 to 10 per cent. of the gas we burn, and it is given off from coke ovens. A very small percentage of it—0·1 per cent.—in the air causes discomfort, headache, and a heaviness of the limbs. It is a danger in the blast furnaces during the smelting of iron, the charging of the furnaces and their tapping, during the manufacture of gas, in cement or brick works, and in tunnel making, while it is the cause of many of the deaths after explosions in coal mines. The inhalation of carbon monoxide produces headache and a loss of power in the legs, and it is this latter symptom that causes the unfortunate miner to fall and be unable to escape into safety when he feels himself being overcome by the gas after an explosion. It is a consolation to have the assurance that the death is quick and painless.

When there is an escape of gas into a room where people are sleeping, the sleep becomes deeper, and if the victims are not rescued in time the sleep passes into death. The dangers of poisoning by gas are not so much in the manufacture of gas as in the uses the gas is put to. A leaky pipe of coal gas is easily detected owing to its strong smell, though it may so saturate the ground as to be a risk to those who are engaged in searching for the leakage. But there is far greater danger attendant on water gas, because it is free from smell, and cannot, therefore, be easily detected.

The Home Office has made various regulations limiting the percentage of carbon monoxide in

water gas to 14 per cent. insisting upon printed bills being posted in factories where this gas is used, warning the workmen of its dangers, compelling the periodical examination of the machinery for leakages, etc., and these precautions are necessary when we find that there were fifty-one cases of poisoning from carbon monoxide gas between 1899 and 1903.

The poison, when it does not kill, still has serious consequences on the nervous system, causing loss of speech, dementia, pains in the limbs, broncho-pneumonia and other symptoms more or less serious.

In the manufacture of waterproof goods and the vulcanisation of india-rubber, a colourless, volatile liquid, called *bisulphide of carbon*, is used. It has an extremely unpleasant odour, but it is also very dangerous when inhaled, causing headache, vomiting and giddiness at first, later a staggering walk, extreme tiredness and loss of appetite. When acting as medical expert on the Dangerous Trades Committee of the Home Office, Sir T. Oliver visited several large india-rubber factories, and found a considerable amount of ill-health among the men and women employed in those departments where bisulphide of carbon is used. Some of the men had suffered for months from paralysis of the arms and legs. Young women are employed in dipping small articles, such as toy balloons and tobacco pouches, into a bath of this liquid, and the effects of the fumes is to cause some of them on their way home to stagger and fall, and it plunges them into a deep sleep from which they wake next morning feeling ill and shaky. It is a curious fact that they continue ill until they have again inhaled a little of the gas.

Others suffer from hysteria, weakening of memory, loss of power in their limbs, difficulty of speech or loss of vision.

Better ventilation and means to draw the draught downwards away from the workers has done much to improve the health in these factories.

There is a great demand nowadays for *quickly drying paints*, for the hurry we all think necessary does not allow us to wait for the usual slow drying of oil paints. These quickly drying paints are not ground with oil, but with petroleum or methylated spirit, or with benzine. It is obvious, therefore, that there is considerable danger to the workmen from the accidental firing of these spirits, but there is also a danger from the inhalation of the vapour. When a ship puts into port the owners insist upon the painting, inside and out, being completed in as short a time as possible, for time is money. Half an hour after the first coat of this paint a second one can be added, but the danger from fire is great. Occasionally the surface of a ship, while being so painted, is enveloped in flames through a spark lighting the vapour given off by the paint. The fumes affect the men more when they are painting the interior of the vessel. They may be overpowered by headache and dizziness and cannot walk steadily, and if they are not quickly rescued the results are very serious. As in all occupations where there is some danger, the workmen should have the risks pointed out to them, besides which there should be no naked lights used, the men should be supervised and should not work for too long at a stretch.

Another industry sometimes causing fire and sometimes poison through inhalation is the dry-cleaning of goods by benzine. The garments to be cleaned are carefully examined to see if the pockets contain matches, and brass buttons are removed to prevent an explosion by friction. Of course no workman is allowed to smoke in the building. In spite of the utmost precautions, fires mysteriously break out in dry-cleaning establishments from time to time, the origin of which has never been explained, and some people believe that benzine has a tendency to spontaneous combustion.

The inhalation of the vapour affects young women more than men, but it is injurious to the health of all, causing hysteria or languor or weakness of the limbs, blindness, neuritis, mental depression and other nervous affections. Recovery may be complete, and it may not. The precautions to be observed in these factories are: all possible safeguards against fire, and means to extinguish a fire should it occur, besides the best ventilation. Also no worker should be allowed to take any food into the workshop.

Under the head of chemical trades we include the manufacture of chloride of lime, bleaching powder, sulphuric and hydrochloric acid.

I cannot describe the process of manufacture of these products in detail; they are generally produced in the same factory.

In the mixing of soda and salt cake (the latter

being used in the manufacture of glass), when common salt is acted upon by oil of vitriol and also when this mixture is raked on to a furnace-bed hydrochloric acid is given off, and the vapour of this has a very irritating effect upon the air-passages of those who inhale it. The workman wears flannel wrapped round his face as a protection. The men's hours are long, and they are exposed to great heat, so that danger from chills is also present.

The manufacture of bleaching powder is unpleasant and dangerous to health, and a man should come out of the chamber, where he is shovelling the powder through holes in the floor into casks underneath, every half hour to breathe the fresh air.

On the whole, chemical workers may be said to be a healthy class until the age of thirty-five, after which the death-rate rises rapidly, and between the ages of forty-five and fifty-five it is twice as high as that of men following an out-door occupation. Diseases of the respiratory system are the commonest cause of death.

In the manufacture of felt hats from rabbit skins the skin is stretched and then rubbed vigorously by men who dip their brushes into a solution of nitrate of mercury, and these workmen suffer from decayed teeth as a consequence, for mercury is a poison, and I shall refer to it in my third lecture.

In the manufacture of jewellery, principally in the refining of precious metals, the workers are sometimes affected by the fumes of nitric acid, generally through the accidental escape of the vapour. The victim of such an accident may not feel the consequences at once, though he may die from the effects within a day or two from acute inflammation of the lungs.

Aniline oil is used in the dying of black alpaca goods, and the fumes given off by its mixture with hydrochloric acid cause illness among the workmen. Cotton goods used to be dyed in this manner, but in consequence of its ill-effects on the workers its use has been almost entirely given up. Similar effects in the workers are frequently caused by the use of para red dyes, a powder obtained from Germany. The men who mix the material in the dye works and those who throw the plastic stuff upon the cloth often suffer from headache, sickness, and they are mildly intoxicated. If they are removed into the fresh air they recover. This para red is used in dying flannelette. I hope you all know what flannelette is. The difference between ordinary calico or cloth and flannelette is that the latter has a nap which is obtained by "raising" one or both surfaces by passing it over revolving rollers. These rollers are provided with steel teeth which draw from the surface a nap, which forms a better non-conducting material for heat than the original cloth, and this, therefore, gives a feeling of warmth to the wearer which the "unraised" fabric does not. Some varieties of flannelette are more inflammable than others, and there is no doubt that the fluffiness of the material causes it to burn with great rapidity, like cotton-wool. According to the 'Lancet' a coroner in 1904 held no less than seventy-three inquests on children who had been burnt to death, and a large proportion of the deaths were due to flannelette igniting. If the poor insist on continuing the use of this warm and cheap material they ought to be prevented from using any flannelette which has not been made non-inflammable.

The manufacture of explosives, which includes gunpowder, nitro-glycerine compounds, ammonium nitrates, detonators and fireworks, is so exceedingly dangerous and can cause such sudden and complete disaster that legislation has had to grapple with the difficulties from early times. The strong arm of the law has interfered, has forbidden all unlicensed manufacture of such materials, and has insisted upon such stringent precautions that this trade, though the most perilous to life, shows a smaller death-rate than many others which are not included under dangerous trades. In the 'Times' of August 25th last, in the annual report of H.M. Inspector of Explosives on the working of the Explosives Act of 1875 during the year 1907, we read:

"In a general opening statement the inspectors say that they have found no reason to complain of any falling off of the general condition of factories and magazines. The occupiers had almost invariably shown perfect readiness to remedy such defects as they pointed out, and the managers themselves, in many instances, introduced improvements tending towards greater safety.

"The number of deaths during the year from accidents by fire and explosion in the manufacture of explosives, *i. e.* six, was below the average for the past ten years, which is 6·9. This number of

deaths was not excessive, and would compare favourably with the death-rate in other dangerous trades.

"Several cases of illegal manufacture of blasting cartridges by miners in their houses have come to light through accidents. Every effort is being made to stop this dangerous practice."

Besides the safety ensured by legislature, there is the fact that the men and women employed in these manufacturies are of a better class, and that the knowledge of the danger of their calling causes them to exercise great care and cleanliness.

It is not in the manufacture of these explosives that accidents are most likely to occur, but in their use, where familiarity seems to breed contempt, and where incredible recklessness is shown by those who use them. During 1899 we are told that there were no less than 29 persons killed in mines, and 195 injured by explosives. When we hear of men scraping out the detonators with a pin, thawing dynamite over a fire, driving dynamite and gunpowder into roughly drilled holes, and boring out misfires, we realise what imminent risks these men run. But besides these accidents there is the effect on miners of the gases liberated during the detonation, the men being in a confined space, that is, in the depths of the mine. They often suffer from headache, vomiting, palpitation and collapse, and only recover when brought into the fresh air. The effects then generally pass off, but occasionally the results are more enduring, the inhalation of these fumes causing a disturbance of the nervous mechanism of the heart.

November 16th, 1908.

THE association of cancer with oak galls was the subject of some interesting comments at the debate on "Heredity with Special Reference to Cancer" at the general meeting of the Fellows of the Royal Society of Medicine on November 11th, and the opportune appearance of Mr. Edward T. Connold's book on 'British Oak Galls,' published by Adlard and Son, London, will serve to satisfy the natural curiosity of scientific men in regard to the principles of oak-gall formation. Several pages of this work are devoted to the interesting and unique features of the cynipidæ, a family of the Hymenoptera. These cynipidæ may correctly be termed gall-wasps and furnish a fascinating study.

PROCIDENTIA UTERI.

A Post-Graduate Lecture delivered at Ancoats' Hospital, Manchester.

By E. STANMORE BISHOP, F.R.C.S.Eng.,
Hon. Surgeon Ancoats Hospital, and Gynæcological Surgeon Jewish Memorial Hospital, Manchester.

PERHAPS no condition is so common in parous women as some amount of descent of the uterus.

It used to be believed that this was usually due to some incautious or unskilful use of instruments, or to some fault on the part of the medical attendant or midwife during the second stage of labour. Any lack of skill or meddlesome midwifery is, of course, to be greatly deprecated, but prolapsus uteri is certainly not due in all cases to anything of this kind. That opinion being held, and because the feeling of weakness could usually be traced to some confinement, many midwives and medical men have, I believe wrongly, been blamed for its occurrence. Such a conclusion is, I believe, often very far from the truth.

And for the following reasons: cases have been observed—I show you one to-day—in which the confinement, which was no doubt rightly credited with the starting of this condition, was entirely over before any attendant whatever saw the mother, where the mischief was done without any outside interference. That is one reason.

And the next arises out of the very anatomical conditions present.

The vaginal canal through which a child has to pass in order to enter the world is one, the walls of which are composed of non-striated muscular fibres, surrounded by a layer of loose erectile tissue, which is most marked towards the vulva (Quain). Now erectile tissue, according to the same authority, contains many elastic fibres.

Clarence Webster, in his last 'Text-book of Diseases of Women,' states that external to Luschka's muscle is connective tissue, containing abundant elastic fibres.

All tubes formed of non-striated muscle and elastic fibres are capable of dilatation without injury up to a certain point, and will return, if that point has not been passed, to their normal size after the departure of the dilating force. The walls of the intestine are similarily composed, and

their dilatability and power of recoil are well known; so is the uterine cervix. But if that point is exceeded, the power of return is impaired and may be entirely, or at least very greatly, lost.

Normally the vagina is only a potential tube, its walls being firmly pressed against one another by the tonicity of the muscular fibres in them, the elasticity of the fibres without, and the tension upon the sides of the levator ani fibres.

You can produce a similar condition much in the same way if you take an elastic ring and pull it lengthways—its sides will straighten out and press against each other.

But in order to obtain that effect the pull must be sufficient in strength and the ring sufficiently small; if the force is too weak or the ring too much enlarged, the latter will remain open.

If an elastic ring is suspended between two immovable supports and a pull is made from these supports upon opposite points of its circumference, the amount of force that it is necessary to exert upon that ring in order to bring it into a straight line will depend, other things being equal, upon the size of the ring, being greater the smaller the ring, since each part of the ring is further from the origin of the force exerted upon it. If in place of a small ring which the force is able to stretch to a straight line, a much larger one is inserted upon which the same force acts, that force will not be able to approximate the anterior and posterior arms of that ring and it will remain open; naturally the same thing would happen with a succession of such rings forming a tube.

If such an elastic tube in the body is distended for not too long a time nor by too large a body, when that body has escaped its natural recoil takes place and it returns to, or near, its former state.

But if the mass be too large or the period of distension too long, the tube, in proportion to these, will lose its power of return to the normal and will remain dilated.

It is easy to see that the valvular character of such a tube will be lost by the mere widening of its calibre consequent upon over-distension.

If, in addition, the tube be also torn below, the curve in it will be still further straightened out, and anything at the upper part of the tube will find it much easier to sink to the bottom.

It is a further peculiarity of the problem we are discussing that whilst such changes are taking place below, other changes involving the increased size and weight of the structure to be kept up have been occurring above.

All these points have their bearing upon the choice of the operation which is to be decided upon in order to restore a prolapsed uterus to its normal position and to retain it there.

The foregoing explanation seems to me more reasonable than that given by Philips in Allbutt's 'Gynæcology.' This writer and others consider that there is a rupture of the fibres of the levator ani.

If you consider that during distension of the vagina the pelvic origins and vaginal insertions of that muscle—the levator ani—are approximated, it is difficult to see how they should be torn; tearing of the vagina in the median line could not affect them since they are inserted into the sides of this tube, but dilatation of the tube itself and their consequent relaxation would put them out of action, and when once the calibre of that tube is restored to its former size we may confidently look to their renewed action to complete our work.

That simple rupture of the not over-distended vagina is not followed by prolapse is proved by cases, of which I have seen three, and which have been frequently recorded, in which complete rupture of the perinæum through the anal sphincter has occurred, whilst the uterus has maintained its normal level.

There are a few cases in which the cervix becomes elongated, so that the os appears at the vulva, whilst the fundus can be felt at its normal level, but those cases are outside the present subject.

The history of the various surgical measures adopted in order to cure the very distressing condition of procidentia is a very interesting one, and reflects step by step the varying opinion of operators as to its causation; it can only very briefly be sketched here.

Naturally, the open gaping orifice through which the uterus protruded attracted attention first, and attempts were made to narrow this so as at least to retain the uterus within the body. Tait's and most other perineorrhaphies are instances of such a proceeding.

By such operations the outer and lower portion of the vaginal tube is narrowed and a shelf is

formed upon which the uterus rests; it can no longer protrude, but it remains at a much lower level than normal.

It must be remembered that with every inch of descent of the uterus from its normal plane an increasing drag is exerted upon its nerves, whilst increasing difficulty is experienced by the blood in its veins and the waste products in its lymphatics in their progress owing to the narrowing and kinking of these tubes. The uterus, in consequence, becomes gorged with blood and lymph, therefore proportionally heavier and more likely to descend still further.

And the use of pessaries, whilst it decreases these later effects, tends to dilate still further the already over-dilated passage. Compare the action of a bougie in the urethra, the mere presence of which is utilised to produce the continuous dilatation there desired.

Next, attention being directed to the heavy uterus which it was desired to lift once more to its proper height, so that the nerve strain might be relieved and the veins and lymphatics once more permitted to pass on their contents at a normal rate, a whole series of operations known variously as ventro-suspensions or ventro-fixations were devised; all these attempt to fix the uterus at a normal or exaggerated height so as to shift the burden from its natural supports to some artificial and unnatural ones.

But the objections to them all are obvious; not only is the new arrangement an artificial one, but any union so made does not last. The gradual effect of the weight of the uterus is to slowly draw out any attachment so made until a band is formed between it and the abdominal wall, beneath which coils of intestine may become strangulated. To avoid this Professor Sinclair and Dr. Wallace, of Liverpool, have lately devised methods which have for their *raison d'être* the desire to obviate any such possibility. Sufficient time has scarcely elapsed in order to show whether these attempts will be ultimately successful; they are no doubt a great improvement upon the earlier and cruder methods.

But even if they avoid such sequelæ entirely, they are still open to the still more serious objections that they do not reproduce in any way the normal state, that by them alone no attempt is made to support the uterus from below by a return to the normal of the parts beneath, that during a succeeding pregnancy a portion of the uterus is hampered in its development, and that such a patient must, in order that such fixation may be carried out, be submitted to an abdominal section, and thereby run the risk, small though it be, of a ventral hernia. I have seen the worst ventral hernia I ever encountered follow an operation for ventro-fixation in a very fat woman.

Alexander's operation, though sometimes advised for this condition, does not really enter this category at all. It is an operation for the cure of retroflexion. It is evident that it can have but little effect in lifting the uterus.

In all plastic operations, here as elsewhere, it is well to keep steadily before the mind's eye the one aim—a return as far as possible to the *status quo ante*—the attempt to replace, as far as can be done, the patient in the condition in which she was before the damage was done.

What, then, was her condition before, and in what respects has it altered?

The uterus was healthy; it was not unduly heavy; its os and cervix were whole.

It was supported by unstretched broad, round, and sacro-uterine ligaments; by a narrow curved vagina, of which the walls were in contact; and by untorn transversi perinei and constrictor vaginæ muscles.

The anal sphincter was intact.

The levator ani muscles were so applied to the walls of the vagina and were at such a tension that they could act at most advantage.

That was the original condition; in what has it altered?

Cases vary in degree; if we take the worst we shall cover all, and anything less will simply mean that certain steps may be omitted as not necessary.

The uterus is in a state of chronic inflammation and engorgement; it is much heavier than normal in consequence; the starting point of its inflammation is a deep tear in the os and cervix, exposing raw surfaces to infection and the internal mucosa, which should be protected, to injurious friction.

Its supporting ligaments have all been unduly stretched and such stretching still continues.

The vagina is unduly dilated and much larger than normal; it is no longer curved but much straighter. Its lax walls, dragged upon by the bladder in front and the rectum behind, which are

still healthily functionating and, therefore, from time to time exerting a peristaltic force, not only on their own structures but on those around, are beginning to sag under the strain.

The transversi perinei and constrictor vaginæ muscles have lost their central attachment and are thereby thrown out of action.

The anal sphincter is torn through.

The levator muscles are shortened and lax, and therefore their force can no longer be exerted.

What surgical steps should be taken to remedy all this?

The ruptured anal sphincter, the transversi perinei, and the constrictor vaginæ must be re-united.

The uterus must be curetted, and to its interior some sufficiently strong antiseptic must be applied, whilst drainage must be allowed for.

The vagina must be narrowed so as once more to allow the levator ani to come into action; by such action a round tube will once more find its anterior and posterior surfaces in contact.

The amount removed must be so planned, both as to amount and position, as that what is left, when re-united, shall form a curve equal to the original vaginal curve.

The manner in which this is done is very difficult to describe in words. I therefore propose to operate before you upon a patient who is suffering in this way.

The first thing that is done, as you see, is the curetting, which must be thorough, then the mopping out of the uterine interior with iodised phenol, the vagina being protected by swabs of cotton-wool soaked in a solution of bicarbonate of soda; when this has been thoroughly done the uterine split is mended by a double Emmett's operation; the rawed surfaces are united by iodised catgut, No. 2, since these sutures must be left to come away of themselves; we cannot endanger the success of the later steps by any later division and removal such as would be required if they were made of silk.

This uterus is not only enlarged, it is also retroflexed; but the fundus is not adherent in Douglas's pouch, for it can be easily brought forward; it will not, however, remain in the normal position, but flops back again when it is no longer held, so something must be done to fix it in the desired position; this is best accomplished by dividing the anterior fornix, separating the bladder from the anterior wall of the uterus, and opening the peritoneum between the two organs. The edge of the peritoneum covering the anterior wall of the uterus is then brought down and fixed with strong catgut sutures to the cut edge of the vagina; the raw surface of the bladder becomes adherent to a portion of the anterior uterine wall above its original site, whilst the attachment of the uterine peritoneum to the anterior wall of the vagina draws forward the fundus and holds it in the desired position whilst that union is effected. The wound in the anterior fornix is closed by sutures, also of catgut, for the same reason as before, in such a manner that what was a transverse incision becomes a vertical one; by this manœuvre the cervix is thrown backwards at the same time that the fundus is drawn forwards. So far, we have restored the uterus to its originally intact condition, rendered its interior free from bacteria, and restored it to its normal slightly anteflexed position; but we have still to arrange for its support at a normal height in the pelvis.

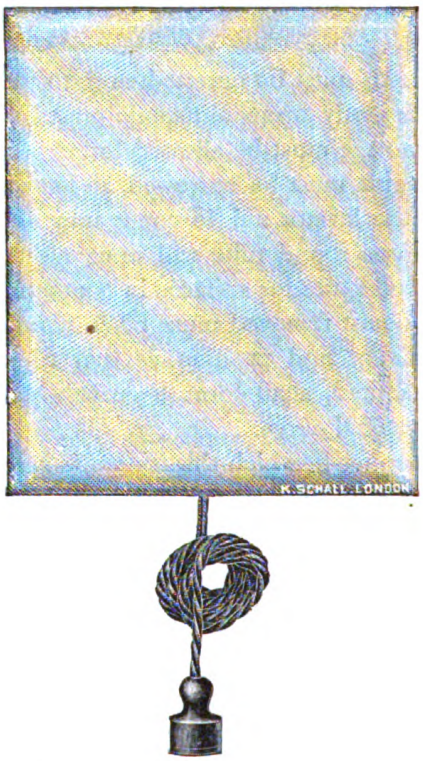
The cervix is slightly drawn forward sufficiently to enable us to grasp a fold of the posterior vaginal wall about half an inch or rather more below it. This fold is divided transversely in the middle line, and from this, as a central and highest point, two incisions are carried downwards and outwards at a wide angle, the width of which will depend upon the amount of relaxation of the walls. As the angle so formed is detached from the tissue between the vaginal wall and the rectum and peritoneal pouch behind, the incised edges in the wall left above are united by strong catgut sutures; the forceps are removed and from this onwards each succeeding suture is made to hold up the tissues and steady them for the insertion of the succeeding stitch; the apex of the flap so removed is held in a pair of Well's forceps and drawn down as it is loosened. When the combined process of division, loosening, and suturing has reached the level of the attachment of the pelvic fascia, the incisions are carried outward at a still wider angle, and these are made to end at a point on either side previously determined upon at the edge of the vulva, and corresponding approximately to the level of the original fourchette. Up to this time the sutures have been of catgut and have merely united the cut edges of the mucosa, but from this

point onwards silkworm gut should be used. Moreover, all the mucosa being removed, the left forefinger of the surgeon is placed in the middle of the wound and is used to depress the rectum and protect it; each stitch is now made to include as much tissue as can be taken up on either side, carried across the rectum, and brought in the same way from below upwards on the opposite side. What was before a raw surface is thus folded upon itself so as to form a central deep scar, which shall unite the separated edges of the pelvic fascia, and approximate the vaginal insertions of all the muscles which originally acted upon it—the levator, the transversales perinei, and the constrictor vaginæ—covering them all and burying them by the united mucosa above. The straight edge of the skin which formed the lower boundary of the vagina is now converted into a **U**, the sides of which are united by fine catgut sutures, and the whole is complete.

This operation was originally devised, I believe, by Dr. Donald, of this city, and whilst it bears evidence of great ingenuity, is practically the most efficient of all the many methods which have been described; for when once the uterus has been placed and held in this way at the normal level, in its normal position, and supported by its normal supports, it may reasonably be expected to lose its engorgement and to decrease in size, since these things were dependent upon the previous mal-position. The stretched ligaments will also, being no longer dragged upon, tend rapidly to recover their original length and tone. And that what we might expect in theory does actually happen in fact is to be seen in these women who have come here to-day to show the results of such operations performed upon them during the past four years.

November 16th, 1908.

We have had submitted for our inspection some examples of Schall's electro-thermal compressors, made for the purpose of treating with dry heat such cases as are suitable for its application. Dry heat, in the shape of radiant heat or electric light baths, is being used so frequently and with such great success that it does not require special recommendation in many cases of cold, and in cases of a rheumatic or gouty nature, such as lumbago, sciatica, neuralgia, gout, and rheumatism. It relieves the pain, reduces swelling, stimulates exudation, and invigorates the circulation. For the relief of lumbago the familiar domestic advice to pass a hot iron over the affected part, protected, of course, by one or two thicknesses of flannel, can be perfectly well justified on medical and scientific grounds, and its undoubted success with well-selected cases in relieving pain bears out the efficacy of the method from a clinical standpoint. Hot air or electric light baths are available in some hydros and special institutes; but in a great many cases it is more convenient and cheaper for treat-



ment to be applied in a patient's own home. The apparatus required is neither costly nor cumbersome, but it can be used only in houses where the electric light is laid on. The electro-thermal compressors consist of strings made of asbestos. Round these strings a fine resistance-wire is wound, and the strings are arranged in spirals between asbestos pads, which are enclosed in an outer cover, which can be removed for cleaning or renewal. The accompanying illustration gives a good idea of one of these compressors. Precaution is taken to place a small rheostat in the circuit to control the heat, which can be raised up to about 300° Fahrenheit as maximum.

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WITH MR. BLAND-SUTTON AT THE MIDDLESEX HOSPITAL.

COCCYGODYNIA.

GENTLEMEN,—I have chosen for my subject to-day a condition of things which is rather slovenly treated in most text-books of surgery, but it is one in which I have taken a great interest: it is coccygodynia, which, if expressed in simple terms, means painful coccyx. My interest in this disease was awakened many years ago when I was quite a lad, and long before it ever occurred to me that I might one day be a surgeon. I picked up an old copy of a periodical containing a lecture on coccygodynia by Sir James Young Simpson, the discoverer of the anæsthetic properties of chloroform, and who, as you will come to learn by-and-bye, was a very distinguished man in obstetrics and in diseases peculiar to women, now termed gynæcology. From that day to this I have always taken a very keen interest in morbid conditions of the coccyx, and especially that form of it known as coccygodynia. Before attempting to describe the disease I shall call your attention to some of the anatomical points connected with that small part of our anatomy, because, small as it is, it is bound up with a number of very interesting questions, morphological, speculative, surgical and pathological. You have all been through the anatomical course, and therefore know quite well that that termination of the spinal column in man and the anthropomorphous apes is extremely rudimentary. As a rule, it consists of three ill-developed vertebral bodies; sometimes there may be as many as five. In the early embryo there are representatives of five coccygeal vertebræ. Usually three coccygeal vertebræ persist, sometimes four, and occasionally five. When the coccyx consists of five vertebræ, its tip will sometimes project a little distance beyond the skin of the buttock and can be easily felt through the skin. The coccyx, as you know,

is the representative in man of the long tails possessed by other mammals: Many cases have been published in the attempt to show that sometimes human beings are born with tails. By-and-bye, when you come to deal with patients, mothers will frequently ask you—it may be after dinner, or it may be in your own consulting room—whether you have ever seen a baby with a tail. From time to time cases are published which are supposed to be examples of human tails. I have spent a very great deal of time in investigating these cases, and yet I tell you frankly that I have never seen a human being with a tail. You will often see children with what are called caudal appendages, but they are not tails. Tails of animals are of two kinds. The tails of horses, or dogs, monkeys, or giraffes contain vertebræ: if you look into the museum and study the anatomy of long-tailed animals you will find that these tails are continuations of the vertebral column. There is another form of tail, which like the pig's tail, contains no bony elements. There are cases in which children are born with a lump sticking out from their buttocks, of which I show you a good example. This is a case which is known as spina bifida. There is a large tumour sticking out from the lower part of the spine, which bad observers might call a tail. It is merely a cystic swelling caused by the bulging of the spinal membranes. Another thing which is often described as a tail is a fœtus with a sacro-coccygeal tumour. Some of these tumours are derived from a portion of the gut which normally extends into the embryonic tail. But in many cases it represents another child attached to a well-formed child, although it is blended together in an ill-shaped mass. When you come to cut and examine such specimens you find that they contain rudiments of another individual; that the man who bears such a tumour is really carrying his brother about with him in the world and habitually sits on him. Later on I shall have to devote a lecture to a consideration of this particular species of tumour, but I want to draw your attention to the fact that the smaller varieties of these tumours are occasionally described as tails, and when the individuals in this condition are born in very poor circumstances of life, you will sometimes see them exhibited as side-shows in public houses, especially in the Whitechapel Road. In those shows these cases are described as children with tails. It is well to go and see these freaks when you have the opportunity; I have myself had many interesting evenings round freak-shows in the Whitechapel Road. Two cases have been published in Germany in which there were children with a long strip-like piece of skin passing off from the sacral region, looking like a pig's tail. But when you come to analyse and look critically into the reported cases of human beings born with tails you will find that they are not so, and that to call them tails is too elastic a use of the term. Most of them are either congenital tumours, which may be sacro-coccygeal tumours, or meningoceles, or fatty tumours. There is an amusing story told relating to a famous old Scotch judge, Lord Monboddo. He believed that all individuals were born with tails, and that it was the duty of the midwife when she attended a lying-in woman to cut off the child's tail. On one occasion whilst Lady Monboddo was adding to the population the midwife heard something moving behind the curtains of the room, and on looking to see what it was she found him hiding and watching with the express intention of seeing her dock the child's tail.

The pelvis of man contains many vestiges of important muscles and structures connected with tails. For instance, if you examine the coccyx with care you will find a ligament running down on its anterior surface connecting it with the sacrum. There is also one on its posterior or dorsal aspect, and two lateral ligaments connecting it with the lower end of the sacrum. They are known as the anterior, posterior, and lateral sacro-coccygeal ligaments. The great sacro-sciatic ligament, which runs from the tuberosity of the ischium to the sides of the sacrum, also sends strong fibres to the sides of the coccyx. Particularly notice this, because it has some bearing on the subject which we shall have to discuss directly, that is, the pain in coccygodynia. We must not lay any stress on this pelvis which I am using to demonstrate these ligaments, because it was made in Paris, and these which you see are not the ligaments at all; they are made of tow and grease and stuck on here to represent true ligaments. The anterior, posterior, and lateral sacro-coccygeal ligaments are remnants of the muscles which in other animals wag the tail. By anatomists these muscles are called the levator caudæ, the depressor caudæ, and the agitator caudæ. The tail muscles

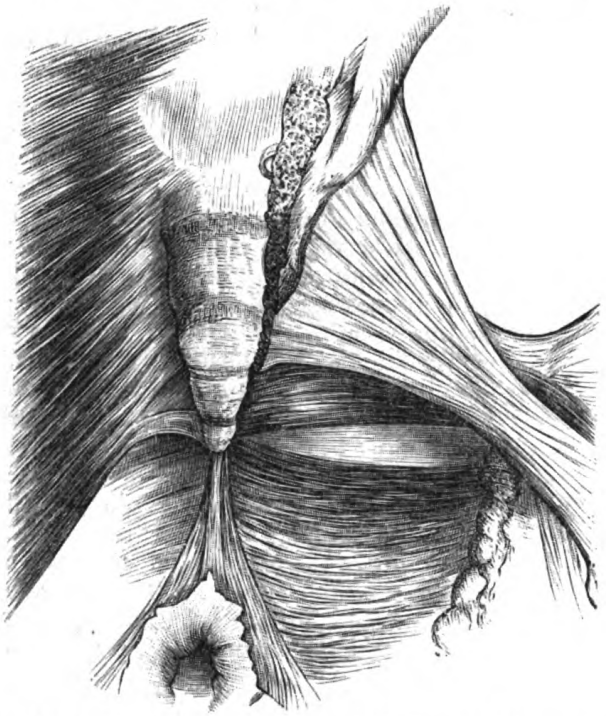
arise from the inner aspect of the pelvis, but when the tail became rudimentary the muscular tissue was gradually replaced by fibrous tissue and became the pelvic fascia, which is so much of a nuisance to you when you begin to study the anatomy of the pelvis. This pelvic fascia is largely derived from the muscles which used to be concerned in moving the tail, and as the tail disappeared so also did the muscles. The coccygeus muscle consists largely of fibrous tissue. If you carefully dissect this great sacro-sciatic ligament you will find that when you come to examine the points of origin of the biceps femoris muscle that its tendon is directly continuous with the fibres of the great sacro-sciatic ligament. If you pull on that tendon firmly you will find it is possible to make the coccyx move. Examples have been observed in which the biceps, instead of arising from the tuberosity of the ischium, are continuous with the great sacro-sciatic ligament, and this ligament occasionally lacks an attachment to the tuberosity of the ischium. This makes it probable that the great sacro-sciatic ligament represents the original origin of the biceps femoris; the tendon has been detached from the muscle by the excessive growth of the tuberosity of the ischium and converted into a ligament. You may probably wonder what this can have to do with diseases of the coccyx.

Although in stout people the coccyx lies deeply buried in the tissues behind the rectum, and you would therefore think that this bone is fairly secure from injury, nevertheless it is often injured, and in a variety of ways. One of the commonest injuries arises at child-birth; it is one of the penalities which women pay who defer getting married until rather late in life, that is to say until thirty-five years of age or after. At that time of life, as you know, the coccyx is very liable to be ankylosed to the end of the sacrum, and it comes to pass that the pressure of the child's head upon that coccyx during delivery will break the coccyx. In some instances the fracture fails to unite and forms a false joint, which becomes extremely painful and produces the condition known as coccygodynia. Another way in which this is broken, in men as well as in women, is by riding. A horse bucks, or the rider has some trouble with the horse, and it bumps him on the saddle, the coccyx comes down on some prominence of the saddle and the bone may be broken. In a number of people it may unite and there is no further trouble. In some individuals it does not unite, or it may unite at a bad angle and cause the patient great pain. Another way in which the accident may happen is by sitting down on the edge of a chair or on a pointed object and so hurt the coccyx. Or you may be at a picnic, and thinking more of the girl you are handing tea to than yourself, and you sit down on a brick or something sharp and break your coccyx. The funniest case of such a fracture which I have seen was that of a man who weighed 18 stone. He had a large house in the country with a broad staircase. He used to often amuse his friends by sitting on a large iron tea-tray and tobogganing down the staircase. One day he slipped off the tray and came down on the edge of a stair and broke his coccyx in three pieces. It was most painful, and at last I had to take the coccyx out. When he had paid the surgeon's fees and the charges of the nursing home he came to the conclusion that it was the most expensive ride for so short a distance he ever took in his life. As I have said, there are various ways in which the coccyx may be fractured. The last patient I operated upon in the hospital was in just before we went for our vacation; she was a gardener's wife, and whilst watching her husband pot plants, she slipped and fell on a flower-pot which was turned upside down. She had great pain in her coccyx until I removed it.

Now a few words as to the muscles attached to the coccyx, then you will understand why it should give rise to so much pain when it is broken. Attached to the side of the coccyx and near its tip there is the levator ani muscle. This muscle contracts spasmodically during defæcation and in powerful deep breathing. The sphincter ani is attached to the tip of the coccyx, and some fibres of the gluteus maximus arise from it, and it affords attachment to a portion of the great sacro-sciatic ligament, which, in certain movements, is pulled upon by the biceps femoris muscle. If you get a typical case of injury of the coccyx, especially where the bone has been broken and pushed in towards the rectum, the symptoms of which the patient will complain are these: First of all these patients complain of pain, often acute pain on the act of sitting, and when they arise from the sitting position. They also complain of pain during defæcation. If you get a patient in this condition you may be sure there is trouble about the coccyx. The pain the patients suffer in the act of sitting is due to the contraction of the gluteus maximus and in some measure to the contraction of the biceps femoris; the contraction of the levator ani and the sphincter ani during defæcation explain the pain attendant on this act.

Now, what are you to do for these cases? The

first thing you do is to try and ascertain whether there is a history of injury. If there is a distinct history of injury, either during delivery or by falls or blows on the coccyx, or anything of that sort, you examine very carefully about the coccyx, and instead of finding it form a graceful curve you will find it tucked at right angles to the spine, and when you introduce your finger into the rectum and find the tip of the coccyx and attempt to press it out again, you produce the pain of which the patient has been complaining. It is said that this pain occurs in the coccyx when there is no history of injury and nothing to account for it, and people say that this kind of pain occurs in hysterical individuals. Whenever a patient complains of



A sketch showing the muscles and ligaments connected with the coccyx. (After Sir James Simpson.)

pain always be very careful before you attribute such a thing to hysteria. Most of these severe pains of which patients complain have got some real cause for them, and when you are not always smart enough at first to ascertain what the cause of the pain is, do not cover your lack of knowledge up by labelling the person as neurotic or hysterical. *Whenever a patient complains of pain in the region of the coccyx, you should examine the anus as well as the coccyx, and satisfy yourself that there is not a painful pile or an anal ulcer.* When you have a patient of this kind who has pain, and you feel the point of the coccyx turned in, and you can feel that through the rectum the movements of the coccyx produce pain, it is worth your while to recommend the patient to have the coccyx removed. That was the recommendation which was made by Sir James Simpson. But he modified his operation in two ways, because in his days even simple operations were attended with risks to life. Any small operation at that date was liable to be followed by suppuration and pyæmia. Therefore surgeons were very careful before submitting a patient to operation. But he tells us that the pain which patients suffered from injury to the coccyx was so severe that even with the risks that there were it was often justifiable to remove the coccyx. But, short of removal of the coccyx, he also suggested in some cases to take a narrow tenotomy knife and introduce it under the skin and go carefully round the coccyx with it, detaching it from all the structures, so that the bone was in that way freed from the muscles. That simple method was sometimes followed by very great relief to the patient. He tells of a case where a lady who hurt her coccyx while riding in India suffered so much pain that she readily submitted herself to the operation. The surgeon with a tenotomy knife detached the coccyx from the muscular structures. He had nearly completed the operation—there was no anæsthesia in those days—when the knife broke in the dense tissues. He said to the patient: "I am sorry my knife has broken and a portion of the blade remains inside." She replied: "Never mind the knife, I am free from pain." The operation can now be made as safe as vaccination. Of course the parts must be prepared antiseptically and the patient laid on her side. Then you make an incision over the coccyx and dissect carefully round in the fibrous tissue, separating the coccyx from it, and taking care not to wound the rectum, which lies immediately beneath it. When you have the sides and tip of the coccyx released, determine exactly the situation of the fracture, and with a knife detach the coccyx at that point. The middle coccygeal artery sometimes bleeds smartly, but is easily controlled. When the bleeding ceases, the soft parts are easily brought into apposition by means of sutures, and the parts are usually soundly healed in twelve days.

Consideration of the coccyx naturally leads me to say a few words about the coccygeal body. That is a little body at or a little beyond the tip of the coccyx. Nobody knows its function, and it is a thing that, when you come to dissect bodies in the ordinary way in the dissecting room, it is so dry that you can scarcely recognise it. But when you are dressers and the surgeon has an opportunity of removing a coccyx, beg it from him, take it to one side and dissect it in the fresh state, and in most instances you will find the coccygeal body.

When coccygodynia was first described it was suggested that probably the pain which patients suffered from may have had something to do with the coccygeal body, but no one seriously entertains this opinion to-day.

November 23rd, 1908.

LEAD POISONING.*

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

WHEN we come to inquire into the various industries in which lead is used, we find that the evil effects of this metal have been known for centuries, and that it has caused more deaths than any other metal, partly because it is so largely used in manufactures, partly because its effects are slow and insidious. The poison lies in the dust, and in the fumes that are inhaled from it, and persons who handle lead compounds or the metal itself are affected by it. During 1907 there were twenty-six deaths from industrial lead poisoning in Great Britain. Lead poisoning is usually easily recognised in its early stages, when it causes colic and is accompanied by a characteristic blue line on the gums, but there are more obscure diseases of the internal organs and of the nervous system which are not always traced to their cause, and which are yet undoubtedly due to the same subtle poison.

It is a pity that lead enters so largely into our common domestic necessities, but when we consider how water is conveyed through lead pipes, how much food is preserved in tins that are frequently contaminated by the lead dissolved out of the solder, how our cooking vessels are enamelled with a lead compound, and our water is stored in lead cisterns we may judge how largely we are subjected to this dangerous metal, and how great a part it plays in the manufactures of this country.

As an instance of the antiquity of lead as a useful metal, I may remind you of the words of Job:

> "Oh that my words were now written!
> Oh that they were inscribed in a book,
> That with an iron pen and lead
> They were graven in the rock for ever."

I will mention a few examples of the dangers to which people of all classes are exposed, even when they are not workers in lead.

You know that the Orleans family, driven from France, found a home in Claremont, near Esher, where they lived for many years. In 1848 some members of the family and several other persons of their household became subject to severe attacks of colic and to some nervous disorders, which their French doctor traced to poisoning by lead. Of thirty-eight residents in Claremont, thirteen were thus affected. It was then found that water had been brought through lead pipes from a distance to a cistern from which the whole household was supplied, and each gallon of water was proved to contain a grain of metallic lead.

Again, some striking instances have been given of lead poisoning from a source which might easily be overlooked—namely, from snuff, falsified by the mixture of red oxide, or sometimes of the yellow chromate of lead.

I have taken this extract from the 'Lancet' of a short time back, showing that lead is still sometimes a danger in drinking water.

"*Lead poisoning at Rochdale.*—At a meeting of the Rochdale Town Council last week, strong complaints were made as to the presence of lead in the water supplied to a portion of the Borough of Rochdale by the Heywood Corporation. Analysis by the Borough Analyst showed that two samples of the water contained one-fifth of a grain of lead per gallon, while a third contained only slightly less than that amount. There were no complaints made of the water supplied from the Rochdale Corporation reservoirs. One of the members of the council said that his son had been suffering for eight months from lead poisoning and a neighbour was also suffering from the same cause. A considerable number of other houses got their water from the same source and new houses were being built and coupled up to the same supply. The council was assured that the Rochdale supply was excellent, and every possible step would be taken to trace the source of the impurity in the water supplied from Heywood."

The lead miner does not suffer from plumbism owing to the fact that the ore contains lead in an almost pure metallic state, but in lead-smelting districts, Leadhills in Scotland and in the Dales of County Durham, the fumes from the furnaces poison the pasturage of the neighbourhood, and the cattle eating this grass have often died from lead poisoning.

The danger to health begins with the smelting of the lead ore. During the year 1906, 38 cases of lead poisoning were reported in smelting works.

* A Lecture delivered at Gresham College, adapted from Sir T. Oliver's writings ('Diseases of Occupation,' 1908, 'Lead Poisoning,' 1891, article in 'Dangerous Trades,' 1902).

If great care be not taken to carry away from the faces of the workmen the fumes given off in the process they are apt to be poisoned. We are told that from each ton of metallic lead which is smelted 130 pounds of lead will be given off as fumes, and it is these fumes from the lead smelting furnaces which poison the pasturage of the neighbourhood. All smelters are therefore subject to lead poisoning, and some families appear to be specially susceptible, one member after another succumbing to it, whilst their comrades in the same work escape.

A very dangerous occupation is the cleaning out of the flues through which the fumes have passed, for the dust is poisonous to man, and when the passages are not freely ventilated the men cannot work in them for more than a couple of hours without suffering from headache and other symptoms. All men employed in this process should have a warm bath immediately after work.

Red-lead workers suffer considerably from plumbism; the metal is roasted in a furnace and occasionally raked. A large amount of fumes escape from the furnace, and unless there are provisions made to carry them away by a strong draught the workmen suffer.

Again, in the grinding of red-lead much dust is raised, which it is dangerous to inhale. In Germany the law enforces the painting of the walls of these factories, so that they can be washed, but in England no such care is taken, and the walls are begrimed with dangerous dust. Young people and all women are specially susceptible to lead poisoning, and they should not be employed in white-lead works, nor in the red-lead furnaces, grinding and packing departments. Sir T. Oliver proves this point by mentioning the following case: "A few years ago at the Klagenfürt white-lead works in Austria, during a scarcity of adult labour, the men begged the employers to allow their sons to work in the factory. Lads under eighteen years of age were consequently given employment. The youths, all of them of good physique, well developed, and healthy looking, were examined medically before entering the factory, and yet, notwithstanding this precaution, so great was the amount of lead poisoning amongst them, and in such a short time too, that the employers were obliged to dismiss them."*

* 'Diseases of Occupation,' p. 144.

No tobacco should ever be allowed in any form in a lead factory, and there should be a special room for the workmen's clothes. In this country the men working in dusty departments wear a special overall, and it would be an advantage if there could be more change of employment from a dangerous department to a healthy one, from inside to outside work. But the greater use of machinery, and therefore the smaller number of hands employed, makes this difficult.

But of all the necessary precautions, the greatest is personal cleanliness. Unfortunately there is a great deal of casual labour in lead works, and in consequence we encounter great poverty and dirt, besides intemperance, which is a special danger in this industry. The casual labourer is less intelligent, more careless, and physically more unsatisfactory, and all factory statistics prove that accidents are fewer when experienced and trained men are employed.

The overalls of the men should be washed once a week, but should be machine washed, for it is a common occurrence for washerwomen so employed to suffer from loss of power in their hands, and from other symptoms of acute lead-poisoning. But above all, cleanliness, hot baths and soap are an absolute necessity, and the industry has certainly become less dangerous of late years since the enforcement of such regulations and the monthly medical examination of the workers.

The British Home Office was the first to notify cases of industrial poisoning, and its organisation is very complete. Legislation and the individual efforts of employers have done much to diminish the number of cases of plumbism in all industries where lead is used, but in spite of these efforts it is a constant source of disappointment to employers and trouble to the workers. It is not large doses of the poison which cause so much and such irretrievable mischief, but long continued, minute quantities absorbed by the system. I referred yesterday to the special tendency towards lead poisoning among men of intemperate habits, and this cannot be too strongly insisted upon. When a man is alcoholic the resistance of his nervous system is enfeebled, and he is often careless as to personal cleanliness, hence his predisposition to this and other forms of poisoning. Very instructive is the instance given by Monsieur Treille, who pointed out in the French Senate the marked

difference between the north and south of France, the intemperate habits of the working men in Normandy and Brittany being well known. He tells us that Normandy alone, with its heavy consumption of alcohol, has more than one half of the total cases of lead paralysis in France.

Another predisposing factor is an empty stomach, and therefore no worker should enter a white-lead factory till he has broken his fast.

I have copied Table I, which has been taken from the annual report of the Chief Inspector of Factories for the year 1906, to show you how bad matters were some years ago, and how Home Office regulations and medical inspection have certainly brought about some improvement.

TABLE I.—*Showing the influence exerted by Home Office Regulations and Medical Inspection on the Prevalence of Lead Poisoning in Great Britain and Ireland.*

Industry.	Number of cases reported.							
	1899	1900	1901	1902	1903	1904	1905	1906
Smelting of metals	61	34	54	28	37	33	24	38
Printing	26	18	23	19	13	15	19	16
File cutting	41	40	46	27	24	20	12	15
Tinning and enamelling	24	16	19	14	18	13	16	18
White lead	399	358	189	143	109	116	90	108
China and earthenware	249	200	106	87	97	106	84	107
Litho transfers	11	10	7	2	3	3	5	5
Glass	8	7	11	8	4	—	3	4
Electric accumulators	32	33	49	16	28	33	27	26
Paints and colours	75	56	56	46	39	32	57	37
Coachmaking	65	70	65	63	74	49	56	85
Shipbuilding	30	32	28	15	24	48	32	26
Paint used in other industries	54	50	61	44	46	27	49	37
Other industries	183	134	149	117	98	102	118	100
Total	1258	1058	863	629	614	597	592	632

In 1898 the Commission appointed by the Home Office to report on white-lead recommended the abolition of female labour in the dangerous processes of white-lead manufacture. There was a great deal of opposition to this, on economic and other grounds, but experience has proved that the Commission was right. And there has been a great diminution in the number of cases since women were excluded from the most dangerous departments. Yet Miss Patterson, His Majesty's Inspector of Factories, tells us that the condition of the female workers in the other departments of the works still calls for constant supervision. Women are extremely susceptible to lead poisoning, and young women often die of acute plumbism a few months after beginning work in the white-lead factories. Besides the influence of sex, young women unfortunately earn lower wages than men, and therefore have to subsist on poorer food and live in worse lodging, both these circumstances making them more prone to disease.

It is a sad fact that women workers, intelligent, practical women, are generally very foolish and careless about providing good food for themselves. I am constantly grappling with the problem how to persuade a woman that if she wants to do good work she must have good food and enough of it. If you want an engine to work you must feed it with good coal; if you neglect to feed it the fire will go out and the engine will sooner or later cease to work. The human body is no more able to work on bad food than is the engine, and if a woman wants to work she must cultivate a healthy appetite and feed as heartily as a man.

Lastly, work in a white lead factory should not be undertaken by any young person. It is fortunate that machinery is replacing hand labour to a great extent.

Before describing to you the symptoms and effects of lead poisoning and then telling you briefly of some of the manufactures where lead is employed, and where the risk from such poisoning is incurred, I will read you a paragraph from Sir T. Oliver's book which is wholly encouraging:

"Formerly Newcastle and Tyneside were hotbeds of industrial lead poisoning. To-day cases of plumbism are few and far between. Until the crusade against lead poisoning there was hardly a week passed without patients thus affected seeking advice at the Royal Infirmary, and scarcely a month went by without there appearing in the daily press the announcement of the death of a lead worker, usually a young female. All this is changed. It is a rare event to have in our infirmary wards a patient suffering from industrial lead poisoning, and especially female in-patients. The declension in the number of cases of industrial plumbism has taken place without a reduction in the total number of persons employed. In 1892 there were forty-four patients admitted into the Royal Infirmary, Newcastle-upon-Tyne, fifteen males and twenty-seven females; in 1900 there

were fourteen patients, all males; since 1900 it has been a rare event to have even one patient the subject of industrial lead poisoning." *

The first symptom which causes the sufferer from lead poisoning to consult a physician is usually colic, but previous to this the patient will tell you that on waking in the morning he has felt headachy and sick, with a peculiar metallic taste in his mouth, while his face has become markedly pale and expressionless. This pallor is due to anæmia, and often rapidly changes a ruddy-faced young girl into a listless, anæmic patient. After headache has persisted for some days or weeks, the patient is often seized quite suddenly with severe pain in the abdomen and vomiting. The pain is so severe that the unfortunate person writhes in agony, and it may last two or three days, gradually subsiding. Death seldom occurs during this stage. It is often difficult for the physician to diagnose these attacks, and they are sometimes mistaken for appendicitis or intestinal obstruction, but a valuable help in the diagnosis is the characteristic blue line along the margin of the gums close to the teeth.

I have referred to the anæmia present in plumbism, and this is due to the decrease of red blood-corpuscles. Now it is the function of the red blood-corpuscles to absorb oxygen from the air in the lungs and to carry this gas into all the organs and tissues of the body. Therefore when they are deficient the system is imperfectly oxygenated, and this has many evil consequences. Lead as a poison attacks the blood-making powers of the body, and it is perhaps for this reason that women cannot tolerate the poison so well as men. Women who suffer from plumbism, even without obvious symptoms, rarely bear a healthy, or even a living child. Women working in a lead factory who have had a series of miscarriages, but who have been persuaded to leave the works and take to an out-door occupation, have then borne healthy offspring. If the women returned to the work they again miscarried. "This loss of offspring is one of the most striking and appalling facts connected with lead poisoning, and is beyond all controversy." When *both* parents are working in lead factories the effects on the offspring are worst, the child when born alive being not more than half the weight of a normal infant, ill-developed and very puny. Next, the infant is most affected if the mother alone is working in lead, but even when the father alone is the lead worker the result to his children is in many instances disastrous.

Salpétrière and Bicètre are very large hospitals in Paris for the treatment of nervous diseases, and one of the physicians, Dr. Roques, speaking of the degenerates found there, says that slowly induced lead poisoning on the part of both parents or in one or other of them is not only the cause of repeated abortion, high percentage of still-births and high death-rate of infants, but is the cause of convulsions, imbecility and idiocy in many of the children who survive the first year of their existence.

In his studies on hereditary degeneration and idiocy, Bourneville places house-painters in the unenviable first rank of the occupations followed by parents begetting mentally weak children.

Lead acts chiefly upon the kidneys, liver and nervous system. The poison may lie latent in the body for years, and then unexpectedly give rise to fresh symptoms. It remains to be seen how the Workmen's Compensation Act will deal with cases of long latent lead poisoning.

The most dangerous form of lead poisoning of the nervous system is cerebral poisoning, to which women are more liable than men. It begins with severe headache, passing into convulsions and coma, and the patient usually dies on the second or third day. If the patient recovers, the vision is often lost. It occurs in young women employed as dipper's assistants in potteries. To quote again:

"On a few occasions I have seen saturnine encephalopathy preceded by seizures which had all the features of ordinary hysteria. A girl comes home from her work, and it is noticed by her friends that she is more excitable than usual. She is inclined to laugh or disposed to cry, and is unable to control either her movements or her feelings. To her friends, and even to medical men who have not had previous experience of such cases, the symptoms do not in any way appear to be serious, and yet within two days after the development of this toxic hysteria the patient will probably be dead." *

The commonest result of lead poisoning, which you have probably all heard of, is "wrist drop." After an attack of colic, when the symptoms have

* 'Diseases of Occupation,' p. 150.

* 'Diseases of Occupation,' p. 206.

yielded to treatment, the patient finds that the muscles of his fingers, hands and wrists become paralysed and hang uselessly by the side of the body, and the unfortunate man is rendered helpless, not only to work, but to feed or dress himself. In severe cases the muscles of the lower extremities and even of the trunk may be affected, and when the muscles used in respiration are attacked the patient dies. Sometimes memory becomes impaired, speech is affected and the paralysis is general, but death is not usual, although it sometimes does occur. Wrist drop may develop suddenly, or it may come very gradually, at first only causing slight pain in the muscles, and when I have seen a young painter with his wrist strapped up with an india-rubber band I have generally supposed that he is trying, quite ineffectually, to divert an attack by inefficient means.

Lead poisoning may be the cause of insanity; and there are many minor evils which it produces, such as swollen or painful joints, painful swelling of the tendons, enlargement of the glands, etc. Sometimes a patient may be cured, but what we should chiefly aim at are preventive measures. The Home Office has issued many regulations for all kinds of lead factories, and if these regulations are loyally carried out there will be much less plumbism in the future; but supervision is necessary to guard against attempts to evade the law. From the experience of those who have lived among lead workers we find that the work-people are apt to be careless and the employers not sufficiently watchful, while medical inspection is neither regular nor complete.

Lead can be swallowed, or it can be inhaled, or it can be absorbed through the skin, or through open wounds. Wherever mechanical apparatus has supplanted hand labour the gain has been great. In the factory of the Union Minière de Bleiberg the number of cases of plumbism fell as a consequence of this substitution from fifty-five to nineteen, over a period of ten years, although the production of lead compounds had been more than doubled; and in the white lead works in Newcastle-on-Tyne equally satisfactory results have followed the introduction of mechanical lifters and carriers.

After factories of white- and red-lead, the trade most productive of lead poisoning is that of china and earthenware. Nine-tenths of the china and earthenware made in England is produced in Staffordshire. It has given more pottery to the world than any other country, and has to struggle hard now to keep its supremacy in the world's market. You probably know that the clay is moulded into the required shape, then placed in an oven and fired. Most ware requires two firings, but before it is put into the oven for the second time it is dipped into a liquid glaze, and this glaze usually contains a large amount of lead It may contain raw lead only, or lead fritted, as it is called, that is, mixed with other compounds. When the ware has been dried after its dipping it is smoothed and cleaned, and if the glaze has contained only raw lead the dust given off in the cleaning is highly injurious to workmen, who must of necessity inhale some of it. Many of the hands suffer from lead poisoning. The glaze on the article melts in the second firing, and, when cool, gives the gloss we see on china and earthenware. About fifty thousand persons are employed in the potteries of Staffordshire, and owing to the reported bad health among them the subject was, about ten years ago, brought to the notice of Parliament, and Professor Thorpe, of the Government Laboratory, and Sir T. Oliver were appointed in 1898 to make a special inquiry into the matter and to ascertain:

(1) How far the danger may be diminished or removed by substituting for the carbonate of lead ordinarily used either (*a*) one or other less soluble compound of lead (*b*) or a leadless glaze.

(2) How far any substitutes found to be harmless or less dangerous than the carbonate lend themselves to the varied practical requirements of the manufacturer.

(3) What other preventive measures can be adopted?

Before reporting, these two experts visited not only the potteries in Staffordshire and Scotland, but also several of the leading manufactories on the Continent, in Belgium, Denmark, Germany and France. Their recommendations finally were as follows:

(1) That by far the greater amount of earthenware of the class already specified, *i. e.* white and cream-coloured ware, can be glazed without the use of lead in any form. It has been demonstrated without the slightest doubt that the ware so made is in no respect inferior to that coated with lead glaze. There seems no reason, therefore, why in the

manufacture of this class of goods the operatives should still continue to be exposed to the evils which the use of lead entails.

(2) There are, however, certain branches of the pottery industry in which it would be more difficult to dispense with the use of lead compounds. But there is no reason why in these cases the lead so employed should not be in the form of a fritted double silicate.

(3) The use of raw lead as an ingredient of glazing material or as an ingredient of colours which have to be subsequently fired should be absolutely prohibited.

(4) As it would be very difficult to insure that innocuous lead glaze should be employed, we are of opinion that young persons and women should be excluded from employment as dippers, dipper's assistants, etc., in factories where lead glaze is used, and that the adult male dippers, etc., should be subjected to systematic medical inspection.

A great deal of opposition arose in consequence of these recommendations, and as the employers and the Home Office could not agree as to how plumbism in the potteries could be diminished without an impossible sacrifice of money, the matter had to be referred to arbitration. It would be, of course, a very impolitic action so to hamper the trade in this country by legislation as to handicap it among foreign competitors.

When the arbitration court met it was found that the employers, perhaps stirred to action by the inquiries and public opinion, had of their own accord done a great deal to improve the methods of manufacture, and they could show that during the two years during which these proceedings had continued, the cases of lead poisoning had fallen from 12 to 3·5 per cent. This result, showing how much might be done through the goodwill of the employers without having recourse to compulsory measures, induced the court to postpone arbitration for eighteen months, to see what further improvements would be made. This adjourned arbitration took place in 1903, and in consequence a series of amended rules was issued by the Home Office. Briefly, these limited the use of lead in glaze, insisted upon a monthly medical examination of workers, the keeping of a health register, the wearing of overalls and head-coverings, and even respirators in certain departments, the ventilation of drying stoves and the daily sprinkling and sweeping of the floors, besides which a scheme of compensation for lead poisoning was arranged.

TABLE II.—*The Number of Cases of Lead Poisoning in the Staffordshire Potteries before Arbitration and since:*

	1899.	1903.	1906.
From the North Staffordshire potteries district . . .	204	75	85
From other districts in the United Kingdom	45	22	22
Total	249	97	107

Table II shows you the number of cases of lead poisoning in potteries before arbitration and since. As you see, there is a marked improvement since 1899, but there are still more cases than there should be, and there has not been the declension in the numbers since 1903 that was hoped for.

The employers have done much to try and improve the welfare of their workpeople, but much more might be done and should be done. There is too much opposition to new and less dangerous methods, too little scientific interest taken in the matter by those most nearly concerned. Recently an exhibition of leadless glaze pottery was held at Church House, Westminster, and those who saw the products were struck by the fact that in spite of all that has been said, the goods were beautiful in colour and workmanship. Many manufacturers persist in saying that they cannot get such depth and beauty of colour without the use of lead in the glaze. Supply follows demand. If the public were aroused to the knowledge of suffering that is caused by lead in pottery works I think they would insist upon having ware made with leadless glaze, even if they had to pay a slightly higher price for it. I often think that if we had more knowledge of the dangers to human life involved in cheap wares (often sweated wares) we should not be so ready to clamour for the cheapest articles.

Another stimulus to the employer may be derived from the Workmen's Compensation Act, under which industrial lead poisoning has been scheduled, so that he may eventually find it cheaper to adopt a healthier process rather than cling to the older methods. The specimens of

leadless glaze pottery I now show you are both inexpensive and beautiful.

Some risk of lead poisoning is run by the men employed in the polishing and cutting of precious stones. It appears that the stone is fixed in a mass of molten metal, containing 60 per cent. of lead, and when this mass is cooled the stone is polished by hand or by an iron wheel. During the fixing of the stone in the lead alloy and during the polishing process the workman runs the risk of being poisoned by lead. There is a considerable number of such cases among the diamond cutters of Amsterdam, where almost all the diamonds are cut. A substitute for the lead alloy has been found in a bed of copper and zinc, but for some unexplained reason the men persist in using the older method in spite of the risk to their health and the suffering many of them go through in consequence. The evil is greatly aggravated where a workman carries on his trade in his own home, filling the atmosphere of the common living-room with lead dust and eating his meals in this atmosphere, often with unwashed hands. Nearly all such workers show symptoms of lead poisoning.

We all know the enamelled iron plates used for advertisements and for the names of railway stations. When these iron plates have been smeared with metallic powder containing often as much as 25 to 35 per cent. of lead, have received their first coating and have been baked and painted, a stencil, with the letters that are to be used cut out of it, is laid on the plate, and women with brushes rub off the surface of the plate exposed through the stencil. Of course a large amount of dust is thereby raised, and the atmosphere of the work-room is thick with dust containing much lead. The workpeople necessarily inhale it, and it produces lead poisoning. The work is generally carried on over perforated tables, with a strong downward draught to draw away the dust from the faces of the workers, but unless great care is taken this draught is not strong enough to prevent mischief. After the stencilling, the plate is again put into the oven and occasionally the process of dusting has to be repeated. Arsenic used to be employed in the enamelling of these iron plates, but it caused so much ill-health that it had to be abandoned. Here, again, the periodical examination of the work-people has done much to improve their health.

When hollow ware is tinned it is dipped in a bath of molten lead and tin, but as the public are eager to get this ware very cheap, less tin and a dangerous amount of lead is used for the cheaper qualities. The man who dips the ware is in imminent danger of lead poisoning unless the pot is well hooded to remove the fumes. On an average eighteen cases of plumbism are reported annually in this trade. But besides the workman, the man who buys the pot and cooks his dinner in it is also in danger of mild plumbism, as the lead in the lining of the pot is dissolved in the process of cooking. In the white enamelling of hollow ware, fortunately, much leadless glaze is now used.

Business in electrical accumulator works is increasing very rapidly, and we still find premises used for the purpose which are quite unsuitable. A good many cases of plumbism are reported from among the men employed in casting, pasting, soldering, trimming, plumbing, and section building, and much of this plumbism is undoubtedly preventable. Here, again, the men should have the dangers of their occupation clearly pointed out to them, should have every facility for warm baths and constant supervision. In Germany a great deal more work of this kind is done, but there seem to be fewer cases of lead poisoning reported, the factories being much more suitable and better equipped.

In 1906 there were sixteen cases of lead poisoning in Great Britain among *printers*, that is the compositors, stereotypers, machine operators, type casters and sorters. The form of poisoning is severe, and the compositor suffers most, probably from the dust caused by the abrasion of the metal (which is an alloy of lead) in his box, and the absorption of lead through the skin when he is handling the type. The metal casting pots should be in separate rooms from the other work, and should have flues to carry away the fumes. The type should be cleaned by steam and the dust sprinkled. In cleaning flues the men should wear respirators. Many printing shops are very ill-ventilated and hot, which should not be the case. But type-setting machines are everywhere replacing the human type-setters; they are more rapid and a saving in human labour. Their use has tended greatly towards the reduction of plumbism among printers.

In the polishing of cut glass and crystal goods, rouge, or putty powder, containing 68 to 70 per

cent. of oxide of lead, mixed with water, is allowed to fall on the revolving brushes, which move at great speed, throwing off a great deal of spray, which falls on the clothes and hands of the workers and on the floor. When this dries it becomes a very poisonous dust, and is inhaled by all those in the workroom. Consequently plumbism exists among them, and many have had to give up the work in consequence of wrist drop and colic. Also, some deaths from cerebral lead poisoning have been reported. A substitute for putty powder should be found, but meanwhile improved methods of polishing and greater cleanliness among the workers have already reduced the number of victims.

In some dye works chromate of lead is used for yellow colours, and in calico printing lead salts are occasionally employed in the printing of colours. The dust given off by the yarn and in the calico drying-room is dangerous, causing plumbism among the workers. The women complain of headache and become anæmic, and occasionally suffer from severe nervous disorders. The following instructive case has been reported:

"In a dye works in which, during the cold weather, the running of the ventilating fan was stopped, there occurred quite an outbreak of lead poisoning. One of the young women unfortunately died from acute plumbism. On re-starting the fan the health of the operatives again improved.*

All dye works should be provided with fans to withdraw the dust from the workers and the people should wear overalls and caps.

Of all the trades which are familar to us, that of painting is the one where we most frequently meet with cases of lead poisoning. Before re-painting a room the old paint is scraped and burnt off and the surfaces are then sand-papered, which causes the workman to inhale much dust. The freshly painted surface gives off emanations which are harmful to many persons, because they contain lead, and there is, undoubtedly, absorption through the skin to be feared by the painter. The inhalation becomes more dangerous inside a confined space. There is no doubt that the white lead used to mix with the oil is very injurious to man. In white paint there is an average of 75 per cent. of lead carbonate and 25 per cent. of oil. You may yourselves have felt headache, lassitude, and a slight nausea after sleeping in a room newly painted, and experiments on animals have shown that they also suffer from these fumes, whilst further experiments proved that when the paint was made with zinc oxide instead of white lead the animal took no harm. It is, unfortunately, not obligatory for house-painters to report cases of lead poisoning, but in spite of this the Home Office had 181 cases notified in 1906, of which 36 died. It has now issued a special form summarising the information under the Factory Act of 1901, which requires medical practitioners to notify cases of industrial poisoning, and the Workmen's Compensation Act now imposes upon the certifying doctor the duty of certifying whether a workman is suffering from a disease mentioned in the schedule, for on the "granting or withholding of such certificate depends (subject to appeal to a medical referee) the power of the workman to claim compensation as if the disease were a personal injury by accident arising out of and in the course of that employment."

Dr. Verhaeghe made an inquiry into the health of the house-painters of Lille. Of 131 he found 68 to be healthy, 45 in fair health, and 18 ill, whilst they suffered most between the ages of thirty and thirty-five. The men suffer from colic, digestive troubles, and wrist drop. The few painters who continue their calling after fifty are those of good habits who have been careful and clean. Long before this age the intemperate men, who suffer first, have dropped out. Here, as in white lead works, we find the baneful influence of the father's profession on his offspring, a large percentage of the children being still-born or dying very young.

Draft regulations have been drawn up by the Home Office for the paint and colour trades, and it is to be hoped that they will have a good influence on those who are employed in the mixing of paints and colours, among whom many cases of lead poisoning occur annually.

The regulations provide for exhaust draughts to carry away the dust, monthly medical examination of the workers, a health register in the factory, the wearing and washing of overalls, provision of washing appliances, and the exclusion of women and young persons in the dangerous departments.

Coach painters, who work in confined spaces, are great sufferers from lead poisoning.

I have not time, nor can I enumerate all the industries where lead is employed, but wherever it enters an industry it brings with it the danger of lead poisoning, and it is only by constant care and watchfulness that the evil to the workers can be avoided.

November 23rd, 1908.

* 'Diseases of Occupation,' p. 182.

THE LENHARTZ TREATMENT OF GASTRIC ULCER.*

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PHYSIOLOGICAL rest for an inflamed structure is an ideal which in many cases is impossible of realisation. The heart must continue to beat, though the valves are studded with vegetations and the pericardium is a bag of pus. The lungs must continue their labours, though many of the alveoli are consolidated, and the pleural membrane is coated with lymph. The kidneys must continue to excrete, though their swollen tubules are engorged with blood. And though active ulceration may be proceeding in the stomach the patient must be fed.

The heart, lungs and kidneys are unable to delegate their duties, but attempts have naturally been made to carry on the process of nutrition by the bowel when the stomach is involved. Yet the considerations here presented may suggest that we have been inclined to under-estimate the powers of the stomach even when diseased, and that even in gastric ulcer nutrition can perhaps be better maintained, and with equal safety, by oral than by rectal feeding.

At the outset we must recognise the limitations of rectal feeding. It is quite impossible to nourish the body at all by putting solid nourishment into the rectum in the form of suppositories. The large intestine is the place where water is absorbed, and our only chance of success is to avail ourselves of this property by giving fluids. It is exceedingly doubtful whether nutrient suppositories are absorbed at all, and I have heard of a case in which nineteen of them were found in the rectum at the post-mortem examination.

Recently the value of nutrient enemata has also been seriously questioned. We may admit at once that the power of the large bowel to digest foodstuffs is very slight. Whether undigested albumen can be dealt with at all is only of theoretical interest; it is certainly absorbed in much too small amounts to make it of any value if thus administered. The only proteolytic ferment secreted by the intestine is erepsin. This ferment, described by Cohnheim in 1903 in the succus entericus, has been shown by Vernon to be present also in all tissues in proportion to their chemical activity, and is probably an important agent in intra-cellular digestion. Its action is to complete the disintegration of proteins beyond the stage of peptones into simpler bodies, in which form they are absorbed. It can also act on caseinogen and fibrin.

That milk has been found the most satisfactory basis for nutrient enemata may be due to the fact that erepsin can digest some of its protein. As erepsin ordinarily acts after pancreatic juice, however, we should naturally pre-digest the proteins by liquor pancreaticus before administering them *per rectum*; this has a further advantage over merely peptonising agents in digesting carbohydrates and fats also. Ten per cent. is believed to be the best concentration for proteins in rectal feeding. As cow's milk contains 4 per cent. of proteins, it would be necessary to add 2 drachms of protein to a 4-ounce enema to bring it up to proper strength. A milk protein, such as protene or plasmon, is a convenient method of doing this.

As carbohydrates are absorbed by the bowel as dextrose, they should be given in this form, and of all the food-stuffs it is claimed that this is utilised best in rectal alimentation. Here, again, 10 per cent. has been recommended, but I have found that in this concentration it is apt to cause some pain, probably because of the osmotic currents it must produce. Fats do not appear to be well absorbed; it is claimed that the fat in the yolk of egg is more readily utilised than other forms of fat, but personally I do not employ eggs in rectal feeding, as they add to the nursing difficulties, already sufficiently great. Should any of the egg be returned it is very offensive.

The nutrient enema which I thought gave me the best results consists of: 4 ounces of milk, 2 drachms of plasmon, 2 drachms of dextrose, 20 grains of bicarbonate of soda, 1 drachm of liquor pancreaticus, 5 minims of tinct. opii. The liquor pancreaticus is allowed to act for twenty minutes at 37° C., and the opium is added just before administration, its purpose being to increase the tolerance of the bowel. The bicarbonate of soda

* Part of a Lecture delivered at the Polyclinic, November 16th, 1908.

is added to imitate the normal alkalinity of the pancreatic juice (1 per cent.). This enema is given every four hours, the rectum being washed out every night and morning by running two ounces of water in and letting it return, repeating the process till a pint has been used altogether.

The total food-stuffs thus given in the twenty-four hours amount to: Proteins, 75 grammes; carbohydrate, 75 grammes; fat, 27 grammes. This is clearly less than the minimum required to keep the body in equilibrium, even supposing that it were all absorbed, which is very far from being the case.

Sharkey* found that as much as 75 per cent. of the solids were absorbed in some of his cases, in which he used sugar, peptones, and pulverised casein. But a possible fallacy in his results is that they were based on the estimation of the nitrogen in the washings from the bowel. That the nitrogen cannot be recovered from the bowel does not prove that it has been utilised by the tissues. That it may be impossible to remove all the administered food is shown by a case related by Herringham.† The patient was a medical man, who was very careful to see that the washings out were properly done. About ten days after first being taken ill he passed an enormous evacuation of the most disgustingly putrid material. And he said: "If I was retaining all that in my intestine for that length of time it is impossible that the treatment can have done me much good."

A more accurate criterion of nutrition is the nitrogenous output in the urine. Laidlaw and Ryffel found that this was approximately the same under rectal feeding as that in the later stages of fasting—as, for instance, in the professional faster Succi, from the fifteenth to the twentieth day. That at best rectal feeding means partial starvation must be admitted; there is usually a progressive loss of weight, and symptoms of acid intoxication may occur. The element of suggestion must not be forgotten in rectal feeding. Even though the amount of absorption may not be large, patients are more content because they think they are being fed. But I have estimated the nitrogenous output in patients* who were merely having normal saline *per rectum*, and in patients on nutrient enemata. It appeared to be the same whichever were employed, and followed the course seen in fasting persons. This strongly suggests that the power of utilising nourishment administered *per rectum* is virtually *nil*. An exception may perhaps be made in favour of dextrose, and I propose to test this point by seeing if liberal quantities of dextrose given thus are able to spare the nitrogenous tissues of the body. But, as I have already said, enemata of dextrose are usually not well borne, as they often cause a good deal of pain.

When one begins to doubt the efficacy of the treatment by rectal feeding, the inconveniences associated with it begin to assume a greater degree of importance. Rectal feeding is disagreeable and sometimes painful to the patient, and it is very difficult to keep him in a cleanly condition. The dryness of the mouth may lead to an ascending parotitis. In these cases I am accustomed to give the patient bismuth lozenges to suck. This serves both to neutralise the gastric juice and to form a protective covering to the ulcer, while keeping the salivary glands active. I am quite sure that this makes it much easier to keep the mouth clean. Incidentally I should like to protest against the use of glycerin in a mouth-wash for this or, indeed, in any other condition. The desiccation which follows only aggravates the state of the mouth. Plain hot water to which a little potassium permanganate has been added is, in my opinion, much to be preferred. Ice is also objectionable; though pleasant at the time, it aggravates thirst.

Starvation, whether partial or complete, cannot be considered an ideal treatment for an anæmic patient who has been recently depleted further by a large hæmorrhage. Moreover, we are now beginning to realise, thanks to Hale White, that hæmatemesis in young women is by no means always due to ulceration, but may be due to a general oozing from the mucous membrane. For this condition he has suggested the name of "gastrostaxis." I was very interested to find that during a period of eight years 428 cases were admitted to the medical wards of St. Bartholomew's Hospital as gastric ulcer. Of these 366 were females and only 62 males. Yet during the same period gastric ulcers were found post-mortem in 20 females and in 22 males. It is very striking to find that the actual mortality was almost exactly equal in the two sexes, while a very large proportion of the successful female cases occurred under thirty. It certainly suggests that in young females we have another cause for hæmatemesis besides ulcer. And if there is no actual ulceration the case against starvation is still stronger.

* 'Lancet,' 1906, vol. ii, p. 1264.

† Herringham, CLINICAL JOURNAL, September 16th, 1908, p. 356.

* Langdon Brown, 'Physiological Principles in Treatment,' Ballière, Tindall & Cox, 1908.

At the Congress for Internal Medicine at Wiesbaden in 1901 Lenhartz protested strongly against the orthodox treatment for gastric ulcer. He urged that in patients who are already in a poor physical condition consequent on one or more hæmorrhages, and indeed often collapsed, the "starvation treatment" not only maintained them in their wretched anæmic state, but might even drag them into a serious state of inanition, which would be very unfavourable to the healing of an ulcer. On the other hand, if enough milk were given by the mouth to maintain the body weight it would overfill the stomach and stretch its walls, thus preventing a contraction of the ulcer and presenting the danger of renewed bleeding.

He maintained that the principles that should guide us in the treatment were as follows:

(i) To furnish nourishment and improve the general condition, and thus favour a more rapid healing.

(ii) To prevent distension of the stomach by limitation of the size of the meals and the amount of fluids; and by the application of ice to the epigastrium.

(iii) To prevent the action of the excess of hydrochloric acid by combining it with food albumen and with bismuth.

These indications were best fulfilled, in his opinion, thus: Feeding by the mouth is started at once, concentrated foods rich in albumen being employed. Food is given at hourly intervals from 7 a.m. to 9 p.m., but complete rest is allowed at night. The patient is fed a tea-spoonful at a time, and is not allowed to feed herself for a fortnight. She is kept in bed for a fortnight, and other medicinal measures, such as the administration of bismuth, can be employed as required.

Essentially the diet consists of iced fresh milk and raw eggs, the whole egg being beaten up and iced. Both milk and egg are prepared in a covered glass tumbler surrounded by ice. The feeding-spoon is also kept iced. Granulated sugar is added to the eggs on the third day; later, raw scraped beef, boiled rice and soaked rusk are also given. The accompanying table gives the quantities allowed in the original Lenhartz dietary; some modifications will be discussed later.

In English measures these quantities mean on the first six days that the patient is taking—

Day.	Eggs (drachms per hour).	Milk (drachms per hour).	Sugar (per diem).	Scraped beef (per diem).
1	2	4	—	—
2	3	6	—	—
3	4	8	1 oz. to eggs	—
4	5	10	1 „ „	—
5	6	12	1½ „ „	—
6	7	14	2 „ „	Rather more than 1 oz. divided into three doses.

No attempt is made to provoke an action of the bowels during the first week at least, to avoid peristalsis, and allow absorption of outpoured blood in the intestines. Then, if necessary, a small glycerine enema or hot-water enema may be used.

The advantages claimed for this treatment are that recovery is more rapid, and that it does not deplete the patient, the food supply being sufficient throughout. The sour regurgitation subsides, vomiting and bleeding stop more quickly, and relapse is less frequent, while pain ceases promptly and morphia is never needed; it is possible to treat the anæmia earlier, and an increase in the body-weight may be manifest as early as the first week.

Before introducing this treatment, Lenhartz noted that recurrent hæmorrhage had occurred in 20 per cent. of the last 100 cases under his care, while in the first 135 cases treated on his plan only 8 per cent. had any recurrence of hæmorrhage. He had only three deaths: one from the severe degree of the anæmia, one from femoral thrombosis leading to pulmonary embolism, and one from the continued erosion of a duodenal ulcer into a vessel; in this case there were five ulcers altogether.

Original Lenhartz Dietary.

Days	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Eggs	2	3	4	5	6	7	8	8	8	8	8	8	8	8
Sugar (with eggs) (in grms.)	—	—	20	20	30	30	40	40	50	50	50	50	50	50
Milk (in c.cs.)	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000	1000
Raw scraped beef (in grms.)	—	—	—	—	—	35	70	70	70	70	70	70	70	70
Milk rice (in grms.)	—	—	—	—	—	—	100	100	200	200	300	300	300	300
Rusk (soaked), 1 piece = 20 grms.	—	—	—	—	—	—	—	1	2	2	3	3	4	5
Raw ham (in grms.)	—	—	—	—	—	—	—	—	—	50	50	50	50	50
Butter (in grms.)	—	—	—	—	—	—	—	—	—	20	40	40	40	40
Represents in calories	280	420	637	777	956	1135	1588	1721	2138	2478	2941	2941	3007	3073

The treatment has been taken up on the continent to a considerable extent, and also in America, but in this country it has received, so far as I am aware, singularly little attention. Haberman * gave a very clear *resumé* of the method, but records of cases treated in this country are scanty. Early in this year Lambert,† in America, insisted on its advantages. The treatment seemed to me well worth a trial, and I have now employed it for nearly a year. I have notes of eleven cases in which I used it, and I have been responsible for its use in several others.

In only two instances did I experience any difficulties with it, and curiously these were both in Jews, who are notoriously difficult to treat. In one of these there was some return of bleeding, but bleeding subsequently returned during the orthodox treatment in this case. In this case the patient objected to every form of treatment, and finally discharged himself. In the other the patient was a very neurotic girl, who objected to this diet, and said that it caused pain; she had some colitis also, and in so far as her objections were genuine it may be that the treatment does not suit this complication.

Apart from these exceptions I have been most favourably impressed by the method, and I am supported in this opinion by my house-physicians and Sisters, who agree that they would much rather have to deal with patients treated on this plan rather than on the other, avoiding, as it does, the misery of starvation and all the discomforts and difficulties of rectal feeding. I was particularly impressed with one case, in a man, æt. 34 years, who was admitted with the history that on each of the three days preceding admission he had had hæmatemesis, losing a very large amount of blood on the first occasion. Immediately after admission he had a fourth attack, but quite a slight one. His medical attendent had seen the former hæmorrhage, and there was melæna after he came in, so there was no doubt as to the reality of the bleeding. He was in an extremely anæmic state, and in the old days would have given me much anxiety if I had had virtually to starve him. The rapid improvement on the Lenhartz diet was most striking. A few beds off was a man who was admitted on the same day for the same condition, but in a less severe degree. He was carefully treated by one of my colleagues on the orthodox plan. The contrast between the comfort and quietude of my patient and the incessant grumbling of the other was as noticeable as the more rapid convalescence on the Lenhartz diet.

It may be thought that there is an inconsistency in my objecting to the use of ice and then advocating a treatment in which ice plays so large a part. The objection to ice, however, applies to those cases that are taking nothing else by mouth. I do not find that patients on the Lenhartz diet complain of thirst.

One important modification I have found advisable to make. Cases in which active ulceration is present usually do not tolerate the raw meat as early as the sixth day, and I do not think it wise to make them try to do so. Pawlow showed that meat extracts were active stimulants to the secretion of acid in the gastric juice, and our object in these cases is to avoid hyperchlorhydria, while fixing such acid as is present by combining it with protein. The nourishment in the 36 grammes of beef can be readily obtained in a less stimulating form by adding 2 drachms of a milk protein such as plasmon or protene to the diet.

Another minor modification of the original dietary, in which I have followed Lambert, is the substitution of cooked minced chicken for the raw ham, which, however suitable for German patients, is not grateful to English palates.

The use of horse-serum has been advocated by Hort * and others for gastric or duodenal ulcer. One of the many functions of serum seems to be the restraint it exerts on the autolytic action of the tissue cells. Now, self-digestion or autolysis goes on more rapidly in fasting than in well-fed tissues. This provides a rational basis for the use of horse-serum in a disease for which most methods of treatment entail more or less starvation. The antipeptic action of serum seems to depend chiefly on the serum-albumen it contains, and Hort has prepared a serum in which this constituent is specially increased in amount. I have employed serum in gastric ulcer, but as it has not been the sole therapeutic agent in any case I am unable to estimate how much benefit was to be attributed to it; but I believe it to be a useful adjunct to treatment, though it is perhaps less called for in cases treated on the plan here recommended than in those treated by rectal feeding. It can be given in daily doses of 30 to 40 c.c., but it must be fresh and sterile, and should be given directly after food when absorption is at its height.

For the conditions of private practice the Lenhartz treatment is particularly suitable. Nutrient enemata require time and skill both in preparation and in administration. I do not think that we can expect them to be given properly except by a trained nurse, whose services cannot always be afforded. But at the best rectal feeding is unpleasant to patient and nurse alike, while its efficacy is open to grave doubts. I would therefore recommend to your careful consideration a method of treatment which is simple, safe, effective, and free from any disagreeable features.

* Haberman, 'Lancet,' 1906, vol. ii, p. 25.
† Lambert, 'Amer. Journ. Med. Sci.,' vol. cxxxv, p. 25.

* Hort, 'Proc. Roy. Soc. Med.,' vol. i, Medical Section, p. 241.

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A CLINICAL LECTURE

ON

SUPRA-PUBIC PROSTATECTOMY.

By W. BRUCE CLARKE, F.R.C.S.,
Surgeon to St. Bartholomew's Hospital.

GENTLEMEN,—Quite recently we have had several cases of enlarged prostate in the wards, on which I have operated. I need, therefore, make no excuse for bringing the subject of enlarged prostate before you in a clinical lecture, more especially as my remarks are based on upwards of a hundred cases on which I have operated, besides others which have been under my care on which no operation has been performed.

It is only within the last few years that anything of the nature of an operation has been attempted for the relief of enlarged prostate. There were, it is true, a few instances in which portions, possibly the whole, of an enlarged prostate was removed, so to speak, by chance when the bladder was being explored for stone or in consequence of severe cystitis, but even this was a very rare occurrence. A prostate which was removed in this way by Mr. Willett is preserved in our museum, and I well remember the patient both before and after operation, and can testify to the great benefit which the operation conferred upon him. After removing a stone from the patient's bladder by lateral lithotomy, practically the only method which was then employed, Mr. Willett encountered a large mass obstructing the neck of the bladder and removed it with his finger. Another piece of prostatic tissue presented after the first mass was removed, and then, if my memory serves me correctly, two or three more pieces followed. The patient made a satisfactory recovery and was able both to hold his water and to pass it at suitable intervals.

Such a method of treatment was, however, looked upon rather as a piece of luck for the patient, and

was not regarded as an operation at all likely to afford relief in cases of enlarged prostate generally.

Surgeons had so long believed that the prostate was too firmly fixed to the base of the bladder to be removed with safety that it needed more than one case, and that an exceptional one, to prove the value of this method of treatment.

But in 1889 a paper was read by MacGill, a Leeds surgeon, at the British Medical Association meeting, which was held that year at Leeds. I was present at the reading of that paper, and I well remember how deep an impression the cases there related made on us, and how eagerly we scanned the bottled prostates which the prostateless patients carried round the room to the amazed audience. From that moment the success of prostatectomy was assured, and but for MacGill's untimely death would no doubt have taken its place amongst the successful operations of surgery sooner than it has done.

The master hand that had designed and carried out the early operations was gone, and, as is often the case, his successors strayed from the principles which he had enunciated, and endeavoured rather to remove the prostate in pieces than to shell it out as a whole, and their success was in inverse ratio to the number of pieces of prostate which they removed. In other words, we had none of us realised that a prostate could be shelled out. *Sometimes it cannot be shelled out.* And when this is the case it is always difficult to remove. Increasing experience has shown us how to get rid both of the hard fibrous prostate as well as of the encapsuled adenoma, but the former class of case must always remain the more difficult and the severer operation of the two. It was, I think, in 1901 that Freyer first published his cases of prostatic enucleation and led us back again to the point in MacGill's path from which we had unwittingly strayed.

Such in a nutshell is the history of supra-pubic prostatectomy, but before I pass on to the symptoms of enlarged prostate and the indications for its treatment by supra-pubic prostatectomy, I must say a word or two on the subject of the removal of this gland by the perinæum. You will remember that the first case, Mr. Willett's, to which I referred in this lecture was removed by that route, and by some surgeons that route is still preferred. Young, of Baltimore, has recorded thirty-eight cases of enlarged prostate on which he has operated by the perinæal route, with a mortality of less than 3 per cent., but I am not aware of any other surgeon who has performed so large a series of cases as this through the perinæum, or who can claim for this method so large a measure of success.

To my mind the supra-pubic operation is destined to supersede that by the perinæum, because it embodies a great surgical principle which must ever make headway, viz., "*See what you are about and don't be content with feeling that which it is quite possible to see.*"

My experience of the surgery of the bladder tells me that all prostatic operations are not simple; that they are occasionally complicated by other conditions of the bladder wall, which it is impossible to feel by the perinæal route, but which can easily be seen, especially with the addition of an electric head light through a supra-pubic opening.

I have more than once come across an unexpected encysted stone and a tumour of the bladder when I have been engaged in the removal of an enlarged prostate. I am absolutely confident that had I adopted the perinæal route instead of the supra-pubic in these cases I should have missed these additional conditions altogether, and my patients would have suffered accordingly.

I need not tell you what an enlarged prostate is, for by this time you will, I am sure, have some idea of the condition to which I am referring. Let us for a moment consider the symptoms which are presented by a case of enlarged prostate. You will see plenty of such cases when you get into practice, and it is well therefore to be on the alert as to their early symptoms.

Your patient will, as a rule, be well over fifty years of age, and in all probability, whatever his age, he will tell you that his symptoms did not start till after he had passed the age of fifty. He probably began to notice about that period of life that he got up at night to pass his water, at first once, and then twice or three times. His calls to urinate during the day were sudden. If he could not find means to empty his bladder, a few drops or a tea-spoonful or two would come away involuntarily.

This nocturnal desire to pass water is by far the commonest and most constant symptom from which these patients suffer, and it may go on for some years before any further trouble is complained of.

Suppose you are consulted by a patient complaining of the above symptoms what steps are you to take in order to make absolutely sure of your diagnosis. Direct your patient to empty his bladder. If his prostate is enlarged it will prevent the whole of the urine from being voided, and that portion which he is unable to pass is spoken of as residual urine. It can only be drawn off with a catheter. Beware of the steps which you take to effect this object. The passage of a catheter under such conditions is often the cause of urinary sepsis. The principles on which asepsis depends are simple, but its successful practice in the surgery of the urinary tract is attended with the very greatest difficulty. Let us reflect for a moment on where the difficulties be, and then we shall stand a better chance of avoiding them. In the first place, the outer half inch or more of the urethra almost always forms a resting place for septic organisms. Assuming, therefore, that your catheter is pure, whatever steps you may take to purify the urethral orifice of your patient you cannot be absolutely certain it is sterile. Beyond this lies the major portion of the urethral canal which *may* contain organisms, and which is practically beyond reach of sterilisation. You see then that the difficulty which besets you is well nigh insuperable. What you can and should do is to take care that your catheter is surgically clean. About this there is no difficulty.

The orifice of the patient's urethra claims your next attention; wash it with soap and water thoroughly before you attempt the passage of a catheter. I say soap and water, because if you employ any of the more powerful germicides you are liable so to irritate the end of the penis as to make future attempts at catheterisation extremely painful and so disagreeable that you may find your persuasive efforts not strong enough to overcome your patient's scruples, and he will perhaps not submit to catheterisation as often as you know it is desirable he should. But even when this is done there is the uncertainty, at any rate of the first half inch of the urethra, in which organisms may be lurking eager to perch on the end of your catheter and get a cheap ride into the interior of the bladder, where they will soon find means of starting septic processes in the residual urine, and the adjacent walls of the bladder with which it is in contact. If septic organisms are thus introduced cystitis will develop, the urine become offensive and contains mucus, pus, and phosphatic and other deposits in abundance. I wish especially to emphasise this state of affairs, because I do not think that the surgeon, whatever may have been his faults in pre-antiseptic days, can fairly be blamed to-day for many of the cases of cystitis which supervene upon enlarged prostate. I have had to treat in the course of my professional career a large number of cases of this kind, some of which were under the care of others before they came to me, and some of which were under my care from the outset; but it is comparatively rare, I am sure, to come across any patient in whom an enlarged prostate has existed for several years who has never suffered from an attack of cystitis, however carefully he has been treated. This fact makes it doubly necessary to consider the propriety of surgical interference in such cases even at an early stage of the disease, and now that the results of operation are so successful early removal is more than ever desirable. My own results for all cases are just under 9 per cent., and if one deducts those cases in which the operation was performed only as a last resource, the mortality at once falls to just over 3 per cent.

The mode of operating.—As a general rule I have employed a general anæsthetic for these patients, but in view of the fact that all such cases must necessarily occur in men who are well advanced in life, I have recently been employing lumbar analgesia, and, as you have seen, with marked success. One great advantage of this method, quite apart from the almost complete absence of shock by which it is accompanied, is the extreme relaxation of the abdominal muscles, and the complete absence of straining on the part of the patient. This enables the surgeon to employ a smaller wound for the purpose, and renders enucleation, which must be performed by the tip of the finger, both easier and less harmful to the adjacent tissues. I need not allude to the exact position in which the patient is placed, as I have quite recently discussed with you this subject at some considerable length. Suffice it only to state that the drug which I find best suited for the purpose is a 5 per cent. solution of novocaine and glucose, and the amount of the solution which is employed is 1·2 c.c. Analgesia begins usually in about three minutes, is complete in five or six minutes, and lasts about an hour.

The bladder is usually washed out and distended with eight to twelve ounces of water before the analgesia is started, in order to save time; but there is no reason why this should not be done after the analgesia is complete, if the surgeon prefers it.

The usual supra-pubic incision, two or three inches in length, is made from the pubes upward, the peritoneum being pushed up out of the way, and the bladder exposed, a metal instrument having been previously passed into it, so as to act as a guide for the surgeon. An incision is made into the bladder and the finger inserted, after which a piece of stout silk is passed through the bladder wall on each side. By this means the bladder can be held up tight against the abdominal wall during the subsequent part of the operation.

The bladder is now explored with the right index finger, and inspected as well if such a procedure is called for. By means of the left hand the prostate is pushed upwards from the perinæum, and if necessary one or two fingers can be introduced into the rectum. Very often, however, the whole operation can be completed without rectal palpation being demanded. As soon as the prostate is steadied between the two hands its removal can be begun. I am accustomed to make a small incision through the mucous membrane in order to give me a starting-point for shelling out the gland, and I have found this plan causes less injury to the mucous membrane than the usual plan of scratching through it with the finger. Experience alone can guide you as to how deep the incision should be made before enucleation is commenced. If the prostate or the large adenomatous tumour, which has practically taken its place, is but loosely connected to the surrounding parts, the process of removal will be easy, and the whole mass may be shelled out in one piece, as you have seen, in less than a minute. If, however, the gland be hard and fibrous, which is happily not often the case, considerable time may be involved in its removal, and in some few instances complete ablation is only possible by picking the gland out piecemeal. The ultimate result of the operation is, however, equally good whether the removal be difficult or easy to perform, but the healing of the wound is usually prolonged in these difficult cases.

The bleeding is sometimes considerable, but it can always be arrested by the application of sponges wrung out in very hot water, with the addition, if necessary, of a small amount of adrenalin.

The bladder is washed out with plenty of water so as to free it from clots, and a large tube inserted for twenty-four or forty-eight hours. It should not be allowed to remain in longer or it will delay the healing of the wound. The bladder should be washed out daily until bleeding has stopped, say for two or three days. It is quite unnecessary, as a rule, to do so any longer. The application of an Irving's apparatus renders the patient far more comfortable, as the urine is conducted away through a rubber tube into a vessel under the bed, instead of soaking into the dressings and necessitating constant change both of dressing and draw sheet.

The question is often asked what becomes of the prostatic urethra? In some cases it is undoubtedly possible to shell out the prostate from behind the urethra and leave it absolutely intact. In my experience, however, this is rarely possible. As a general rule the prostatic urethra is removed with the gland, and may be seen, as I have often pointed out, *in situ* when the prostate is examined after it has been removed. In this case the prostatic urethra is separated from the membranous portion where those two parts of the urethra join, and the subsequent history of these cases indicates that micturition is in no wise interfered with by its removal. There is, I have no doubt, some tendency for the cut end of the membranous urethra to close up if steps are not taken during the recovery from the operation to pass an instrument, preferably a metal one, from time to time. I have never in my own cases come across such a condition, and I attribute it to the fact that I have always taken this precaution, but I have had two patients brought to me for subsequent treatment whose prostates have been removed, and in whom the urethra was completely blocked. In both these cases I was obliged to re-open the supra-pubic wound, which had been reduced to a mere fistulous tract, and then succeeded only after considerable trouble in introducing a metal instrument, which had to be pushed through the blind end of the urethra into the bladder. Both these patients eventually succeeded in passing their water the right way, but their period of recovery was much prolonged.

You may, perhaps, be surprised that I have not so far alluded to the time-honoured custom of rectal examination in cases of prostatic enlargement. I have purposely not done so because in most instances it is absolutely useless. What you want to determine is how far the urinary outflow is obstructed, and this can only be discovered by the passage of a catheter. The size of the prostate is no gauge of its obstructive power. I have known a prostate to be so large on the rectal aspect that it demanded colotomy in order to prevent obstruction. This was before the days of prostatic removal. Nowadays no doubt a gland of this size would be successfully removed through the bladder. Even a prostate of this magnitude did not interfere with micturition, because it did not happen to encroach upon the urethra. Remember, therefore, that though you may easily feel *per rectum* whether the prostate is enlarged, you can never determine by this method whether the urinary outlet is interfered with, and that is what you want to know.

November 30th, 1908.

POISONING FROM SOME METALS AND FROM DUST.*

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

WE spoke last night of the effects of lead poisoning, and we must now pass on to other forms of metallic poisoning, beginning with arsenic.

In the form of green arsenite of copper, arsenic was at one time used as a colour in wall-papers, and as a fine dust the arsenic used to flake off, affecting those living in the room.

One of my earliest recollections is that my sister and other children became very ill while staying at the seaside. Two died, and it was not discovered until the survivors were removed from the house that the illness was due to a green wall-paper containing arsenic.

The first fatal case of arsenical poisoning from wall-papers occurred in 1858, but before that date many of those employed in the trade had suffered from ill-health. In those days the paper would sometimes contain as much as 59 per cent. of arsenite of copper. In 1877 the Medical Society of London reported upon this, and lead and arsenic are now no longer used in the colouring of wall-papers. Since this has been accomplished that trade is no longer the unhealthy one it was. Vegetable colouring material is used and is quite harmless. Unfortunately, during the process of receiving its colour the workpeople are living in a very hot atmosphere, and there is some dust given off by the paper, which, like all dust, is bad to inhale.

Personal cleanliness of the workpeople, baths, the wearing of overalls, and the use of fans have contributed largely to the improved health in these factories.

Men employed in arsenic works are affected by the fumes, and also by the rapid changes of temperature to which they are submitted. But the most important fact to be noted is that arsenic, either taken as a medicine, inhaled in fumes or applied to the skin, appears to predispose the tissues to cancerous growths.

* A Lecture delivered at Gresham College, and partly adapted from Sir Thomas Oliver's 'Diseases of Occupation.'

Arsenic is used in curing certain furs, probably as a destroyer of insect life. By American law one grain per square yard is allowed, but some specimens of furs lately examined by American chemists revealed the fact that the number of grains per square yard often reached 170. I need hardly say that such a large quantity of arsenic in an article that is to be worn is exceedingly injurious. Some years ago a few cases of arsenic poisoning occurred in this country which were traced to a particular muslin worn by the victims.

Copper does not, as a rule, unpleasantly affect the workmen who have to deal with it, although the vegetation in the neighbourhood of copper-smelting works suffers, but occasionally copper-workers do complain of symptoms of dizziness, nervous depression and staggering gait. Metallic copper is less harmful than some of its compounds, such as brass. Sometimes food cooked in a copper saucepan will cause intestinal derangement, and therefore enamelled saucepans are chiefly used in England, but abroad, where copper is still in vogue, great care must be taken.

Brass is an alloy of zinc and copper. During the pouring of the hot molten alloy into moulds a dense white smoke rises into the atmosphere. This smoke contains oxide of zinc. When the workshops are not well ventilated the workmen complain of headache, sore throat, and digestive troubles, but the illness they are specially subject to is what is popularly called "brass-founder's ague," which has no connection whatever with real ague or malaria, though the symptoms—headache, shivering, nausea, and a rise of temperature—somewhat resemble it.

The dusty atmosphere the men work in also tends to give them bronchitis and other chest troubles. The preventive measures against such ill-health are personal cleanliness, good ventilation, proper means for the escape of the fumes and for the removal of the dust. Women and young people under eighteen are not allowed to work in the casting shop, where the danger is greatest.

Mercury is used in making barometers, thermometers and other scientific instruments, also in the arts, in the separation of gold and silver from their respective ores, in the manufacture of incandescent lamps, in bronzing the interior of field-glasses and other industries. Mercury, whether absorbed or inhaled, is a poison, and no man with

an open sore on his body should be allowed to work with this metal. Formerly it was used in the silvering of mirrors, and caused much illness among the workmen, but it is now not used for this purpose.

The poison causes ulceration of the gums, loss of teeth, muscular tremors and debility.

Many years ago the mercury mines of Almaden in Spain caused such ill-health among the workmen that convicts were imported to do the labour. The unfortunate men not only worked but lived in the mine, their habits were filthy, and the mortality from mercurial poisoning was in consequence very high. The few free men employed divided their time between the mines and agricultural work, and as they were also more cleanly in their habits they had, on the whole, good health.

In the extraction of gold from its ore mercury plays an important part, but the resources of science have lately found a substitute for mercury, and there is consequently an improvement in the health of the workers.

Mercury becomes volatile at a low temperature, and it is this fact which causes it to be so dangerous to the workmen, who inhale the vapour given off by the metal. They become pale and suffer from giddiness and headache, while the muscles of their face, limbs and tongue become tremulous, the gums swell and ulcerate and the teeth become loose, speech is slow and difficult, the patient loses the power to walk and is depressed and melancholy. Some are seized with convulsions, others with paralysis. With this poison, as in the case of lead, the work of the parents affects the children, who are ill-nourished and rickety and specially subject to phthisis.

Cases of mercurial poisoning are notifiable to the Home Office. The precautions to be observed by the workers are: scrupulous personal cleanliness, the wearing of overalls and respirators, good ventilation, and periodical medical examination, which is provided at the factories.

We now come to consider the dangers attendant on the manufacture of iron and steel. Iron is seldom found in such a state of purity but that it requires roasting in large furnaces to get rid of foreign material. The temperature is raised to a great height so as to allow fusion of the metal to take place, and it is then run off in a molten form into pits of sand. The iron, when cooled, has the shape given it in the sand beds, and is called "pig-iron." In my first lecture I spoke of the dangers incurred by the men who charge the furnaces, who are liable to inhale carbon monoxide gas. These men are exposed to sudden and great changes of temperature, their work is very hard, and they consequently run the risks of chills, rheumatism and bronchitis, besides being the victims of accidents. The pig bars are again heated in a furnace while they undergo the process of "puddling," that is, being rolled about by means of long iron tools, to get rid of carbon. This work is extremely hard. The men are exposed to terrific heat, and the muscular exertion required of them is great, so that they perspire freely and drink freely. There is a severe strain on the heart which often causes heart disease, and the risk from accidents and burns is always present. The puddler's work is undoubtedly one of the most trying in iron factories. But steel is everywhere replacing finished iron, as we see in ship building and other industries, so that the puddler's occupation is a dying industry, while the manufacture of steel is growing apace.

The risks in iron and steel foundries are mostly from accidents and the effects of burns. The finished steel is poured into moulds, during which explosions may occur. Although we have few statistics to show what particular diseases our steel-workers are subject to, German figures show that the death-rate from phthisis among them in that country is high, probably owing to the dust inhaled.

A common complaint among ironworkers and boiler-makers is deafness, caused by the great shock to the ears from the loud noises of the hammers. Cotton-wool in the ears modifies the noise somewhat.

Among workers in iron we may include blacksmiths. Machinery is rapidly taking the place of handwork where heavy hammering is required, but the blacksmith's work is still arduous, and induces heart affections and some forms of paralysis. One would think the occupation was a very healthy one, but a considerable number of blacksmiths suffer from tuberculosis, and this again must be due to the inhalation of dust. No doubt a more muscular type of man formerly adopted this trade, for the machinery now used makes it possible for a less

robust individual to take it up, but this is a pity. The work still requires very considerable physical strength, and where this is not present the blacksmith suffers from rupture of the muscles of his arm, besides heart affections and chills.

We now come to *steel grinding*, which includes the vexed question of dry and wet grinding. Sheffield is, of course, the home of this industry, and I have lately had the opportunity of visiting it, thanks to the hospitality of the Master Cutler. But for generations this industry has been notorious for its high death-rate from lung diseases. Let me first explain to those who do not know how the grinding is done. It is skilled labour for which a period of apprenticeship has to be served. The grinding is done on a circular stone wheel driven by steam or water power. This wheel has to be carefully chosen and tested, because it is run at a terrific speed, and the slightest flaw may cause it to burst, the fragments flying in all directions and causing death or injury to the workmen in the vicinity. The grinder sits astride the grinding stone on a saddle, cramped and doubled up, with his arms pressing the blade of the instrument he is grinding down towards the stone. It is, of course, a position in which the lungs are compressed, so that they cannot be filled, and the air is full of particles from the grinding stone and the steel instrument.

There are two ways of grinding cutlery, the wet and the dry method, the dry method being by far the more dangerous. The dust, raised by the wheel while the metal is pressed on it, is in the form of very fine powder, and must of necessity be inhaled by the man at work. Forks and needles, we are told, are generally ground by the dry method, knives, scissors, and razors by the wet.

In wet grinding there is a trough of water below the stone through which it revolves, so that the surface is always wet and hardly any dust is given off.

Where there is dry grinding fans are provided to carry off the dust, but unfortunately men grow careless over the work they are too familiar with, and often do not trouble to see whether the fan is working or not. Mr. Edmund Owen referred to this in a popular lecture delivered at the annual meeting of the British Medical Association held at Sheffield in July last, and said: "If each particle of dust in a dry grinding shed could be shown as a tiny barb, which indeed it is, and could for once be seen as such by the workman, I think that he would be more inclined to take advantage of every arrangement which the Home Office has designed and the masters have made for his greater safety.

"At the beginning of last century very little dry grinding was done. The sheds were placed along the side of the Don for the sake of the wheel power. Thus there was plenty of water close at hand, and it was made to drip on the wheels. And we are informed by the late Dr. Arlidge that when the Sheffield workers were exposed all day long to winds and draughts and cold their health was better than when they had taken up their quarters in closed factories. Every master and every man at that time was turning out only one kind of work—the best. Those were the good days before 'cheap' cutlery was made.

"As soon as it was found that the wheels could be worked by steam power, the river-side sheds were deserted. The men and boys came crowding into factories and workshops, and found themselves able to do much more work. Indeed, so busy were they that they ceased to moisten their wheels. They lived in an atmosphere of crowd, bustle, noise and dust. Truly they were making more money, but this was often at the sacrifice of health. When they came into the factories they lost the breezes which, blowing in on every side, had kept the air of their old sheds wholesome, and, as many of them became less robust, they began to think that fresh, cold air might be positively harmful. I dare say that at the present day there are Sheffielders who look upon an open window on a February day much as the Devil looks upon holy water; yet open windows and fresh air become more than ever necessary as people crowd together. They should keep the windows of their workshops open, sleep with their bedroom window open, and accustom themselves to the thought that fresh air is their best friend, and one for which no duty has to be paid. In the first part of the last century the windows of our houses were so heavily taxed that poor people were often compelled to shut out much of their light and air. It was only in 1851 that this tax was taken off, and no Chancellor of the Exchequer would ever again think of resorting to it, however short he might be of money. It is probable that he would even prefer to arrange an import duty

on French motor cars, German pianos, Swiss grinding organs, and American cutlery.

"One cannot, of course, go back to the river-side sheds, nor take away bodily the walls of the factories, but one should not be afraid of fresh air, either in a workshop, a dwelling-house, or a railway carriage. I am not going to say that wet grinding on a chilly day in February is likely to be a pleasant occupation. I fancy that sitting over a wet wheel for so many hours a day in a damp shed with a muddy floor would give me lumbago; but, at any rate, I should have the satisfaction of knowing that I was not filling the air with dust to damage my own lungs or those of my fellow-workers—which is something. And if I may venture to look into the future, I will say that when next the British Medical Association meets at Sheffield, dry grinding will be spoken of as a thing of the past, much as we at the present time speak of rushlights, of tinder-boxes, and of flint-lock muskets."

Now what is the result of dust created by dry grinding? So long ago as 1865 Dr. Hall, of Sheffield, drew attention to the high death-rate of steel grinders from consumption. It was a terrible fact that at this period the average age at death of steel grinders was only 29. Matters have improved since then. In Sheffield now chest diseases of all kinds account for 64 per cent. of the deaths among steel-grinders, whereas the percentage among other adults is only 32, therefore, roughly speaking, steel workers are twice as liable to consumption, asthma, pneumonia, pleurisy and bronchitis as other men. In Dr. Hall's time, as I have said, the average age of the steel-grinder at death was 29, at present it is 43.

In the report of the Factory Inspector for the North East Division for this year attention is drawn to the interesting statement of Dr. Scurfield, Medical Officer of Health at Sheffield, in regard to the dusty trades of that city. A comparison is drawn between the death-rate from consumption per thousand for steel-grinders in Solingen, which is the Sheffield of Germany, and the death-rate from the same disease among grinders in Sheffield. The latter is considerably higher. In 1905 the death-rate at Solingen among the steel-grinders was nearly 8, in Sheffield 14.

There are estimated to be 29,000 persons employed in the making of all kinds of cutlery, knives, forks, scissors and swords at Solingen. Consumption was found to be so prevalent among them, as compared with men working in other trades, that the attention of the Government Factory Department was attracted to it. With characteristic energy and thoroughness measures were at once devised to counteract the evil. The factories at Solingen are now said to be marvels of cleanliness and order. The floors are of concrete, and are swept clean every evening and damp-wiped once a week. The walls of the workrooms are lime-washed every year and the air space for each worker is 565 cubic feet. The atmosphere of the town is clear, owing to the use of coal briquettes and to careful firing in the factories, the grinding stones are protected by guards to prevent the danger of their bursting, and as a result of all this care and attention to detail the death-rate is rapidly and most satisfactorily declining.

Since it has been demonstrated that the reduced death-rate in Solingen has followed improvements introduced into the trade, more especially in regard to the methods of collecting and extracting dust, there seems no reason why, with the adoption of similar methods, the stigma attaching to the dusty trades of Sheffield should not also be removed.

About thirty years ago the emery wheel was introduced into Sheffield for the shaping of knife handles, and as these handles are held together by steel rivets large quantities of steel and emery dust are given off during the grinding process. The wheels are made of crushed emery stone with some binding substance, and they revolve at a very great pace. Suction should always be provided to draw the dust downwards and away from the face of the worker, and there is some risk attached to the fact that occasionally a wheel breaks, scattering fragments to a great distance and causing much injury. It is said that the emery wheel has been the cause of even more ill health among cutlers than before its introduction. The workmen suffer from intense thirst and sore throat, but after a rest from their work for eight or ten days the symptoms usually disappear.

I have had occasion to refer again and again to the harmful effects of dusty occupations, and it will be as well for me here to explain shortly the injury that is done to man by this universal enemy. All dust, be it remembered, is harmful, though

some dust is much more injurious than others. It is a growing evil in these days of motor cars, and one that has to be faced. When you speak to a farmer he will tell you that the hay fields bordering popular high roads, where the motors are rushing by all day, now yield a crop not worth half the money they were formerly valued at, because the dust has so smothered the grass that it is stunted and dry, and the grit in it is most harmful to the animals who eat it.

Here is an instance of the mischief caused by trade dust, quoted by Mr. Owen: "Some years ago a physician brought before a society of medical men in London pieces of the diseased lungs from a man who, for many years, had been a razor grinder. Nearly all that time he had been worried by asthma, but he lived by his work at his dry wheel, and he had to put up with it. As likely as not there were other dry grinders in the same shed, and so the air was full of dust from stone and steel. And this dust proved to be very harmful to him; for the pieces of lung which the doctor showed, instead of being light and spongy, as they should have been, were heavy and solid, and on carefully looking at thin slices of them with a magnifying glass glistening cystals could be seen, which, on further examination, turned out to be tiny particles which had flown off from the stone as the man bent hour after hour over his work. Doubtless his lungs were weak to begin with, and the dust with which he and his fellow-workers filled the air got down into his tubes, made him miserable by asthma, and paved the way for a fatal attack of pneumonia."

*Table showing the Mortality from Phthisis and other Diseases of the Respiratory System in different Dusty Occupations.**

Occupation.	Comparative mortality figures (all causes).	Mortality figures.	
		Phthisis.	Diseases of the respiratory system.
Agriculturist	602	106	115
Potter (earthenware manufacture)	1706	333	668
Cutler	1516	382	518
File maker	1810	402	423
Glass maker	1487	295	445
Copper worker	1381	294	406
Iron and steel manufacture	1301	195	450
Stone quarrier	1176	269	307
Brass worker	1088	279	273
Chimney-sweep	1311	260	291
Lead worker	1783	148	397
Cotton manufacture	1141	202	338

* Taken from Sir T. Oliver's 'Dangerous Trades.'

From the above table you will see how, in certain industries, the mortality from consumption and other diseases of the respiratory system is from three to four times as high as among the agricultural class. The agriculturist is in some ways specially privileged, for he has not only little dust to contend with, but he also leads an open-air life in the pure country, but, as you see, specially dusty trades, when the dust is of a dangerous type, have the highest rate of mortality.

The question of the health of the underground railway worker must depend considerably upon the quality of the air he breathes. Dr. Soper, of New York, has published a report upon the air and the dust in the Rapid Transit Railroad, which runs for twenty-one miles under that city. The air was found to contain almost the same amount of oxygen as the outside air, the amount of carbonic acid was not large, there were only half as many bacteria, and the microbes found in the subway were, presumably, those of the streets, for they varied as those in the streets did, and were more numerous in the underground near the stairways. The dust which was carried down the stairs contained more micro-organisms than the dust of the subway, and as these are often harmful to man it is important to note that the absence of sunlight in the subway allowed them to live a great deal longer. Thus the pneumococci lived in the subway for twenty-one days, but only four days in the street.

The dust of the underground railway was found to contain more than 61 per cent. of iron, which is due to the grinding action of the breaks on the wheels and lines. This, of course, is very injurious to man. But no serious disease appears to be prevalent among the New York underground railway workers, though they suffer from catarrh of the nose, throat and windpipe, and also from dry pleurisy.

Thirty years ago the deaths from consumption among cotton spinners were much more numerous than they are now, but even to-day there are more than there should be. The Medical Officer of

Health for Blackburn tells us that the conditions under which cotton operatives are working are better now than at any previous time in the history of the industry, and, as a consequence, the mortality rate has diminished, the health of the operatives has been better, and there has been a larger output of manufactured goods.

The principal factors that contribute to the ill-health in cotton mills are fouling of the air breathed by the workers by gas, dust and emanation of the soil, excessive humidity in the weaving sheds, high temperature and want of cleanliness. In 1889 the Cotton Cloth Factories Act was passed, insisting upon sufficient cubic feet of air per person, and prohibiting the manufacturers exceeding certain limits of moisture. It is difficult and injurious to people to work in damp air such as was considered necessary in the weaving sheds; the clothes of the workpeople became saturated with moisture, which led to chills, rheumatism and colds; and damp air is enervating and unfits the worker for fatigue, because the evaporation of the skin is interfered with, and thereby one of the natural methods of cooling the body is prevented.

The regulations have generally worked well, and the health of the operatives is fairly satisfactory.

Although the Home Office has done much in recent years to improve the health of the workers in the linen factories of Belfast, Lurgen, and Lisburne, there is still some ill-health owing to certain conditions of the workrooms. The work is hard, and the rooms are kept at a very high temperature, from 90° to 125° F., while some of the rooms are too moist and others too dusty. There are complaints of dry sore throats in the dusty departments, where the rough flax is converted into tow. The men employed in doing this are called "hecklers" in the trade. While in the spinning-room the workers complain of headache and giddiness owing to the heat. Others have to stand all day on wet floors, and they may suffer from varicose veins and foot troubles, and those first entering the mills often have a peculiar fever with nausea, vomiting and headache, owing to the disagreeable smell of the oil and the stifling atmosphere.

The jute industry in this country gives employment to about 40,000 persons, and almost all of it is made in Dundee. Jute is a vegetable fibre and is exported from India in bales. It is used to make sacks and coarse twine. Part of the process of manufacture is extremely dirty and dusty, and exhaust fans have to be in constant use, but in spite of these fans the people employed in these departments, *i. e.* those who prepare and spin the fibre, suffer from the effects of the inhalation, and many cases of bronchitis and other chest troubles, including pneumonia, come into the Dundee Infirmary every year. The factories are exceedingly noisy, and owing partly to this, partly to the dust in the moist air, which tends to form plugs of wax in the ear, there is much deafness among the jute workers. These people are very poor, and their housing is very imperfect. Their rooms are overcrowded, and there is much female labour employed in the factories. All these facts may be the reason why the children of these workers are exceptionally puny and ill developed.

It is a sad fact, and one which calls for redress through the strength of public opinion, that wherever women are employed in factories the children inevitably suffer. No woman can work all day and attend to her home duties also. To these mothers baking, washing, cooking, and sewing are dead arts. They confide their children—the weakly offspring of overworked parents—to the care of paid help, and this is done as a matter of course. Their mothers did the same with them, and those who survive will repeat the process with their children. It is not always an economic gain, for the women do not earn high wages and have to pay to put their children out to nurse. Thus, a certain woman in a Yorkshire factory admitted that she earned ten shillings a week but had to spend twelve shillings for the care of her home.

Factory workers obviously cannot nurse their offspring, with the usual result of a high rate of mortality among the infants. The children are hand-fed by ignorant hirelings, who drug them to silence their wailing. In a deputation which waited upon Mr. Asquith to urge greater legislative restriction of the employment of mothers in factories, the following fact is mentioned: "During the Lancashire cotton famine, while privation increased the actual death-rate, the infant death-rate was greatly lessened owing to the mothers being compelled to suckle their infants."* And writing

* 'Dangerous Trades,' p. 75.

of the effect in Coventry of the prostration of trade in 1861, the Registrar-General says: "The care of the mothers of Coventry has, it would seem, counteracted some of the effects of privation, so that neglect of their homes by mothers at work in factories is apparently more fatal than starvation."*

Mrs. Tennant, formerly Superintending Inspector of Factories, writes thus of infantile mortality and factory labour: "On the one hand is the infant death-rate steadily rising, on the other the birth-rate steadily falling; and to the rising death-rate maternal neglect gives impetus, while the State inattentively takes note. But though statistical speech is eloquent of death, it says nothing of withering injury to life; nothing of injury to the mother who, herself affected in health, produces less fitting children; nothing of those children who escape death to endure life, crippled in body and in mind, drugged and starved in infancy, neglected in childhood. What dare we ask of their womanhood and their manhood? What service of citzenship have we any right to demand, any reason to expect? We must seek our answer from within our reformatories and our asylums, from our hospitals, our workhouses, and our prisons. For the feeble in body and dwarfed in mind there is no room in the struggle for industrial supremacy. Their drifting place is among criminals, in the lowest ranks of industry, or in the homes of charity." †

Poisoning by African boxwood is one of the industrial diseases recommended for compensation by the Workmen's Compensation Act Committee of the Home Office. The shuttles used for weaving purposes are made from this wood, which causes various symptoms in the men engaged in work upon it, making them pale and jaundiced, while others have attacks of difficulty in breathing. It is during the sandpapering process that a fine dust is inhaled from the wood which produces indisposition. Other wood used for rulers and also teak wood cause ill-effects among the workers.

One would imagine that rag-sorters would be an unhealthy class, nor can we think of any more distasteful occupation than that of sorting the rags collected from all parts of the world in every condition of filth. But it is not the case. Rag-sorters are generally women, and their work consists in sorting the material into two kinds—(1) cotton and linen rags which are made into paper, and (2) woollen rags that are converted into cloth. All bundles of rags ought to be disinfected before they are opened, partly to destroy parasites and partly to ensure against any septic poisoning or infectious illnesses. When the bundles of rags are first opened the dust from them is injurious, and later on, when the rags are ground by machinery or beaten, the process causes a terrible amount of foul dust. New hands at the business suffer from what is popularly called "shoddy fever," with a rise of temperature, severe headache and catarrh, the symptoms resembling an attack of influenza. All workers in the shoddy factories should be re-vaccinated against smallpox, and there should be good ventilation and cleanliness on the part of the workers.

A large percentage of chimney-sweeps are the victims of cancer, and this can only be explained by the fact that the soot of coal has a peculiarly irritating character, the irritant being by some supposed to be arsenic. Dr. Tatham gives as the mortality figures from cancer 156 for chimney-sweeps compared with 44 for men engaged in other occupations. A gardener who was in the habit of using soot to sprinkle upon plants to protect them from slugs suffered from cancer of the hand employed. Although cancer is the principal danger in a chimney-sweep's calling, he is also very liable to affections of the lungs, due, of course, to the inhalation of irritating particles of soot. Even in these days, when chimney-sweeps no longer climb chimneys, it must be recognised as a dangerous profession.

I understand that there are now in England only five men capable of climbing chimneys, all dating from the time when apprentices at a tender age were employed by hard taskmasters. One of them lately came to my house and climbed a chimney in order to remedy a fault. He told me that he had been climbing chimneys since the age of nine, and that it was impossible to learn the art of twisting the body into the chimney recesses after that age. He had been apprenticed to his uncle, and I naturally asked him if he had had any personal experience of cruelty before the Acts of Parliament were passed at the instigation of Lord Shaftesbury. He said that on one occasion a

* *Ibid.*, p. 75.
† *Ibid.*, p. 74.

master sweep for whom he was working lighted some straw under him to compel him to mount more quickly, but that he had at once descended and attacked his master, who promised never to repeat it and kept his promise.

When we come to investigate the condition of tin-plate workers we find a very unsatisfactory state of affairs. In the tinning of cannisters and iron boxes the iron is dipped into a bath of molten tin to obtain a coating of lustrous white. The industry is mostly confined to Wales. The works are practically in the open air, which would be good if the workpeople—chiefly women and girls—were not put at a serious disadvantage by being constantly soaked during the washing of the plates in a specially prepared bath. We hear that it is impossible for them to keep their skirts, underclothing, and boots dry. His Majesty's Inspector of Factories, Miss Squire, gives a sorrowful account of the condition of these women. She says that, besides the women who are wet during the washing of the plates, "an equally wet, unwholesome, and somewhat degrading occupation is that of the girls who have to carry the iron plates to and from the pickling bath, in which the plates are dipped in sulphuric acid solution. The girls are literally soaked through to the skin by this acid solution, which destroys their clothes. The fumes given off by the sulphuric acid are unpleasantly strong and pervade the whole of the works. For the removal of the fumes and the steam the workpeople rely upon the draughts through the mill dispersing them. The workpeople suffer from inflammatory affections of the eyes, accompanied by a discharge, dryness of the throat, nausea and giddiness; the teeth become black in consequence of the sulphuric acid fumes, and the hair falls off. Occasionally the girls wear a handkerchief over their mouth as a protection." Besides these objections, which are bad enough, we hear that the burdens the young girls have to carry are often far beyond their strength. In the tin-plate works of South Wales it is not uncommon to find girls carrying iron plates weighing from 40 to 100 lb.

In a review of the annual report of the Chief Inspector of Factories for last year we read: "It might be thought that, after a century of factory legislation in this country, it would be impossible to find evidence of women and girls. but especially the latter, carrying heavy weights far beyond their strength. Such, however, is not the case, judging from the reports of the lady inspectors, whose statements almost take us back to the days of child slavery in brickyards. Only by prosecuting offenders and by inflicting heavy penalties upon them will it be possible to put an end to a system which is as cruel as it is unnecessary."

November 30th, 1908.

A CASE IN WHICH A PORTION OF A LUNG WAS EXCISED.

Delivered before the Medical Society of London.

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And Discussion thereon by

Dr. Charles Chapman.
Dr. F. de Havilland Hall.
Mr. Kellock.
Dr. Clifford Beale.
Dr. Heywood Smith.
Dr. Kingston Fowler.
Mr. A. E. Barker.
Dr. Samuel West.
Mr. Lockwood.

The patient was a married woman, æt. 36 years. In the early part of July, 1906, she consulted a dentist. "Chloroform" was administered by a surgeon and a considerable number (six or eight) of stumps were extracted from both jaws. On recovering from the anæsthetic she discovered a stump lying loose between her cheek and gum and spat it out. At the same time she felt difficulty in breathing and a tightness in her chest, and felt sure that a "stump had gone down her throat."

From the first she was troubled with a cough, though previously she had been quite well, and a few days later noticed a pain in her chest, chiefly under the breast bone. Three weeks later she began to expectorate a thick, tenacious, green sputum. In October, 1906, she had a bad sore throat, lost her voice, and spat blood along with the thick sputum for fourteen days. In December, 1906, she again spat blood, but only for one day. In January, 1907, her tonsils were removed with the object, she states, of removing the cough. Shortly after the pain returned in her chest. In March, 1907, she was very ill, weak, coughing badly, and bringing up a profuse fœtid expectoration, not blood-stained. At this time she was entirely confined to bed for three weeks, and was an invalid for ten weeks. She gradually improved and went away for a change for seven weeks, and though her cough did not leave she got stronger. In September, 1907, she again spat blood, and a bad pain appeared in her left side. In December, 1907, she was so ill that she was again confined to bed, expectorating profusely a horribly offensive tenacious fluid and feeling very weak. Slow improvement followed

till April, 1908, when the cough got worse; pain was felt in both sides of her chest, and the fœtid expectoration increased.

I saw her first on May 25th, 1908, nearly two years after the dental operation, from which she dated the commencement of her illness.

She was sent by an eminent practitioner with the diagnosis of "a tooth impacted in a bronchus." She looked pale and weak, was frequently worried by a cough, and expectorated with difficulty a quantity (about one pint in twenty-four hours) of most offensive greenish-white fluid. The same day she was admitted into Dr. Drummond's ward in the Royal Victoria Infirmary for observation and treatment, and there she stayed till June 22nd (one month).

The diagnosis made by Dr. Drummond was that she had bronchiectasis limited to the lower part of the left lung, and though Röntgen ray examination showed no tooth, both of us believed from knowledge of similar cases that the mischief was due to an impacted tooth. The prognosis made by Dr. Drummond was that she could not get better, and he entirely approved of my suggestion that the lower part of the lung should be excised. One hour before operation the patient was placed in such a position, by raising the foot of the bed, that her head was down and her feet up, and in this position she was encouraged to empty her bronchi by forcible coughing. When this had been accomplished, $\frac{1}{4}$ gr. of morphia and $\frac{1}{100}$ gr. of atropine were administered subcutaneously.

At the operation on June 27th, 1908, chloroform was administered by Vernon Harcourt's inhaler, and the patient was turned over to the prone position with her left chest anteriorly pressing upon the table and lowermost, and her right chest uppermost and supported underneath by a pillow, the object of this being to limit mechanically the chest movements on the left (diseased side). A large curved incision, commencing close to the outer edge of the sternum and ending just beyond the posterior axillary line, convex downwards, was made over the lower part of the chest, and a flap, including all the soft parts covering the ribs, was reflected upwards. A small opening was now made into the pleura through which a needle, such as is used for transfusion, was introduced. During the succeeding stages of the operation, between one and a half and two pints of normal saline (3j of salt to one pint of water), at a temperature of 105° F., was run slowly into the pleural cavity. After resection of 6 in. of the eighth rib the pleura was widely opened and most of the saline escaped. It was then found that the lower part of the lung was fixed by universal adhesions to the diaphragm, which were not so firm as to make its separation difficult. At this stage, but for firm pleural adhesions in the groove at the side of the spine, it would have been easy to pull out the lower lobe of the lung and complete the operation for its excision. Dense adhesions, which required some cutting for their separation, prevented this, and it was necessary to remove portions of the seventh and sixth ribs to gain freer access and to bring the adhesions into view. After their separation the pleural cavity was packed with gauze all round the portion of lung to be excised. This was then drawn out, clamped across with forceps, divided beyond the forceps in four different portions, and excised without loss of blood further than what escaped from the parietes and adhesions. After cleansing the divided lung surface the lower part of the pleura was filled by gauze wrung out of saline and dusted with boracic powder, the gauze being so arranged as to prevent contact of the cut surface of lung or of the clamps with the pleural cavity, and to receive any discharge that might occur. The wound was left widely open with the clamps and gauze ends projecting from it, all being covered by a voluminous dressing. At the end of the operation the patient was in good condition, and but little shock followed it.

The after-progress of the case was as follows: Continuous rectal infusion of normal saline was used from the first. On the day following operation she was found lying on her left side and looking no worse than patients do after an ordinary abdominal operation. The outside dressings were frequently changed as there was considerable serous oozing from the pleural cavity. Three days after the operation the clamps were removed, leaving the gauze *in situ*. Five days after the operation the gauze was removed, the lower pleural cavity was cleansed, and fresh gauze was reinserted. During the first week she had no cough, and felt and looked wonderfully well. Eight days after the operation her temperature began to rise, and she had some cough, but this never became a trouble-

some symptom as it had been before. Eleven days after the operation she looked ill and flushed, had a temperature of 103° F., and pulse 118. Respiration 38. It was suspected then that she had pneumonia. Eighteen days after the operation her temperature had fallen to normal, pulse 106, and though her respirations were 40 she looked ill. From this time she steadily went down hill, with high temperature and difficult breathing, and died somewhat suddenly twenty-six days after operation.

Post-mortem examination showed at the bottom of the wound in the chest-wall a cavity the size of an orange. The wound and the cavity in the pleura were lined with healthy, clean granulation tissue. All the open bronchi, with one exception, had completely closed and were invisible. The single exception was plugged with mucus, and on introducing a probe into this at a distance of three quarters of an inch from the cut surface a foreign body which felt like a tooth was struck. That this was the missing stump can be seen in the cut specimen. The remainder of the pleural cavity was filled by the lung, which was everywhere fixed to the parietal pleura by recent lymph. Both lungs were otherwise healthy. The pericardium, visceral and parietal, was plastered over with purulent lymph. There was nothing abnormal elsewhere. It was obvious that death resulted from pericarditis.

Dr. CHARLES CHAPMAN asked whether the treatment of inverting the patient in the early period of the illness had been tried. He was curious to know what a vigorous smack on the back would have done for such a patient.

Dr. DE HAVILLAND HALL desired to draw attention to the recent improvements by W. Brüning in Killian's apparatus for investigating the larynx. By this means it was possible to see into the trachea, and even into the bronchi and some of their branches, and to remove foreign bodies from quite low down in the lung. That afternoon, at Westminster Hospital, at the invitation of Mr. Stonham, Mr. Tilley gave a very interesting demonstration as to the ease with which the procedure could be carried out, and Mr. Tilley exhibited a pin which he had extracted from one of the divisions of the bronchi by means of the apparatus. Therefore he, Dr. Hall, thought that in future those distressing cases of foreign bodies becoming impacted in the lung, and causing bronchiectasis and gangrene of the lung, should become less frequent, because the apparatus did not seem to him to require any very great amount of skill, no more than could be readily acquired by any ordinary practising surgeon. Of course it would have been too late to use such an instrument at the time the patient under discussion consulted Mr. Morison, but when patients had had the misfortune to have a foreign body impacted in the lung they would be only too ready to apply to a surgeon if they felt there was a prospect of it being removed.

Mr. KELLOCK asked whether Mr. Morison could account for the fact that the X rays failed to reveal the tooth. He would have thought that even in the centre of the lung a foreign body of that nature would have been revealed by the rays.

Dr. CLIFFORD BEALE also mentioned a case of missing tooth, which came rather prominently forward a short time ago. In that case there was very decided dyspnœa, but it was quite uncertain whether it was due to obstruction up above or below. But examination was at once made with the X-ray apparatus, and it was seen that the tooth was above the larynx, in the base of the epiglottis, from which position it was easily removed. Most practitioners must have seen cases of foreign bodies in the lungs, and as most of them led to gangrene with fatal results, the question of removal by operation was an important one. But a case which was under observation for many years came under his notice again recently. The patient was a lady who got some rabbit bone down into the air-passages, and it was never found exactly where it had gone to. She was very ill at the time, but made some sort of recovery, and then got into a chronic condition, in which she periodically brought up considerable quantities of offensive pus. It was at that time she was sent to him with the object of finding out whether it was possible to relieve her. The condition had clearly become a chronic abscess of the lung. The X-rays showed plainly where the trouble was, and the question arose as to what should be done. He had a consultation with a surgeon who was not wanting in boldness and did his best, exploring the chest as thoroughly as possible. But the abscess cavity could not be hit so as to bring off the smallest quantity of pus.

Eventually there was nothing for it but to leave the case and do all that was possible by means of hygiene, etc. The patient lived a very careful life, spending much of it in the open air, and it could now be seen that the abscess cavity was gradually getting smaller.

Dr. HEYWOOD SMITH said perhaps Mr. Morison would be able to say why it was, considering the patient had a suspicion of a tooth being in the lung or in the chest, that X-ray examination did not afford more information. It was known that fluid was a hindrance to the passage of the X rays, and possibly the amount of discharge round the tooth might have masked it in that way. And he would like to hear why the whole case was not earlier investigated by the rays.

Dr. KINGSTON FOWLER said he remembered a case very similar to the present one, but in which no operation was done. The case was under the care of the late Mr. Marshall, who was surgeon to Brompton Hospital. The tooth occupied exactly the same position as in this case. An empyema formed, and the cavity in the lung was opened, an attempt being made to dislodge the tooth. The patient was in the hospital on several occasions, and ultimately developed pulmonary tuberculosis and died. He could not look at the present specimen without being exceedingly sorry that an attempt which was so nearly successful in removing the whole of the disease should not have been completely so, and that the foreign body should have just been missed. He thought it was the most remarkable specimen of bronchiectasis that had ever been removed by surgical operation. If the operation was ever to be attended with success it must be in such a case as the present one. With regard to the other specimen, one could see that the whole of the disease had practically been removed except that part which immediately surrounded the impacted tooth. He could not think there could be a large future before operations for removal of the lung. Mr. Morison was fortunate in finding adhesions at the base, but one could never be sure of the presence of adhesions. It had been said that a man could diagnose a pleuritic effusion, but it would be admitted that that was certainly very difficult. Bronchiectasis following impaction of a tooth might be more diffuse than in the present case, not merely limited to one lobe. If the condition lasted a long time it was likely to have extended to the opposite lobe. It was very unfortunate that the tooth was not included in the portion of lung removed at the operation, but he thought a tooth generally lodged at that particular spot, and if subsequent cases of the kind should be operated upon, the lesson learnt by the present case would probably not be lost sight of.

Mr. A. E. BARKER asked whether Mr. Morison or some of the surgeons or physicians present could see whether there were many cases on record where, after the formation of an empyema, the tooth had been discharged externally without operation. If there were many cases of the kind one would be disposed to wait until the abscess or collection of matter had approached more within one's reach. He could not recall such a case in which such a collection of matter had been opened and yet the tooth had been discharged. He had been very much tempted to do such an operation this year on a case in which a tooth had been lodged in the right bronchus, but he was very glad he did not attempt it. The patient's condition was very serious at the time, and the operation would have had to be a severe one, and he thought the operation would have led to the commencement of an empyema. A large empyema did form and was aspirated ten days after he saw the patient, and after that an attempt was made to remove the tooth, but the first few whiffs of chloroform were fatal.

Dr. SAMUEL WEST said there were many instances in which foreign bodies had been expelled after a long time, but they were generally of such a kind that they could be easily brought up. If the foreign body were such a thing as a jagged tooth that was not so. There had been some remarkable operations performed. One was recently carried out for a foreign body in the right bronchus in which the chest was opened from the front, and a flap of the chest wall was turned back. The lung collapsed, and it was moved aside until the bronchus was reached; the foreign body was then successfully extracted. The chest was then washed out and stitched up without any regard to the air which the pleura contained. It was a most successful case, and the patient made a complete recovery. The prospects of operative success depend on where the foreign body is located. If it is in the bronchus or trachea one might have some idea

where to go, and how to deal with it. When it came to being lodged deep in the lung, where it was difficult to obtain exact information as to where it was located, operation must after all be guess-work, and he always felt a good deal of compunction in sanctioning surgery which was guess-work, because his experience was that it was not successful. There was the same difficulty in connection with localised empyemata. It was all very well to operate if one knew where the foreign body was. If there was a difficulty in knowing exactly what was present it was wiser not to open the chest. He thought Mr. Morison was to be congratulated on the success of the operation. Half an inch higher would have made the operation a triumph. It was interesting to know that large portions of the lung could be removed with success, and if Mr. Morison had gone higher it might have been a great triumph. But only in a limited number of cases were operations on the lung possible. He did not think it was justifiable to operate in cases of phthisis, because it was impossible to remove the whole disease, and there were very few things except foreign bodies which would justify operations on the lung.

The PRESIDENT said it seemed very hard for Mr. Morison to have been so near success and yet to have failed. He wondered why none of the surgeons present had referred to the surgical technique pursued. Mr. Morison evidently used his clamps because he was afraid of hæmorrhage from the cut surface of the lung. He questioned whether cutting the periphery of the lung was likely to be attended with serious hæmorrhage. A little while ago he removed a portion of lung at the periphery, and he did it as easily as removing a portion of the periphery of the liver, there was little bleeding, and what bleeding there was was controlled by smearing over with the cautery. He was sure Mr. Morison must feel pleased at the result of having afforded such an interesting discussion.

Mr. MORISON, in reply, thanked the meeting for its reception of his paper. He did not know why the X rays did not show the tooth. At the post-mortem examination the rays showed the tooth quite plainly. The Rontgen-ray apparatus at the hospital was in the hands of a very skilled man, in whom they had great confindence, and that operator did not know why the tooth did not show in the living body. Dr. Kingston Fowler remarked that he thought he was fortunate in meeting with adhesions. But he (Mr. Morison) thought that was a misfortune; he did not think the adhesions helped him at all. He filled the pleura with saline fluid so as to do away with the need of the artificial help of adhesions, to make a pseudo-empyema of it, and he could easily have removed the portion of lung which contained the tooth if there had been no adhesions. But the adhesions were so tough that he was glad to get the operation finished. With regard to Dr. West's remark concerning guesswork, it was guesswork about the tooth, but no guesswork about the bronchiectasis, and the operation was undertaken for the cure of the latter. He was assured by his corresponding physician that the condition was an incurable one, that it was likely to spread, and that it was at present localised. Therefore he (Mr. Morison) said those reasons sufficed to influence him in favour of surgery, and the operation was undertaken for the removal of the bronchiectasis, and not for the removal of the tooth, because they were not certain as to that.

November 30th, 1908.

Cæsarean Section.—Pfannenstiel says that the indications for Cæsarean section should be much widened in view of the good results obtained with the operation with modern surgical technique. It ought to take the place of operations involving the bony pelvis whenever the condition of the patient is such as to call for as short a post-operative stay in bed as possible. In most cases the patient, after a Cæsarean section, may be allowed to sit up in bed three or four days after the operation, especially if the Pfannenstiel abdominal incision has been made use of. Cæsarean section could also be performed with advantage in dangerous conditions calling for an immediate emptying of the uterus; eclampsia and acute nephritis may be counted among such cases if any difficulty may be expected with the more usual methods of quick delivery of the patient. In cases of living child in a transverse position with threatening rupture of the uterus both the child and the mother may be saved by a properly performed Cæsarean section.—*Medical Record*, vol. lxxiv, No. 19.

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WITH DR. F. J. SMITH AT THE LONDON HOSPITAL:

ON SOME DISEASES AND THEIR MANAGEMENT.

IN the management and treatment of disease two therapeutic measures must at once be admitted to stand head and shoulders above all others combined, whether these two be considered from a quantitative or qualitative point of view; they are, bed or rest, and food, for quantitatively, do we not put nearly every patient (certainly all who are acutely ill) to bed, and almost without exception we make suggestions as to the food of those under our care, either because we think it advisable or because the patient wishes for our guidance on the matter. Qualitatively, I have no hesitation in asserting that by a timely application of enforced rest in bed, or *per contra* by permissive or compulsory extraction of a patient from bed, by a timely restriction in diet, or *per contra* an encouragement to a fuller diet, an unlimited and incalculable number of disasters are avoided far greater than those avoided by a similar timely exhibition of drugs, however specific they may be in their action.

Out of the bacteriological studies of the last quarter of a century has arisen a prevalent fashion of springing upon the profession new methods of treatment by inoculations, vaccines, etc., based upon experimental research into the properties of various sera. I would be the last in the world to deprecate such studies on the part of our scientific bacteriologists, believing, as I do, that they have bestowed on mankind many invaluable means of treatment, and are destined to give us in the future a complete key to therapy when disease is attributable to microbic influence; but I cannot help but feel that the premature publication of one-sided results of such studies has a tendency to

lead medical men away from the study of their patients as living creatures, to lead them to neglect clinical observation, and to try to substitute for such observation the fascinating certainties of scientific deduction, which too often, alas, are not certainties at all, else surely we should have been spared the infliction of a recent paper attributing to horse serum the relief of symptoms in gastric ulcer, when it has been a commonplace of clinical observation for years that the same relief comes from simple rest in bed and sloppy diet. Hence it is that I offer you no apology for the fact that my lecture deals with no new therapeutical measures, that the reasons for my opening statements are more of a clinical than experimental character, and based much more on a somewhat long experience of patients and thoughtful observation than on scientific data. Who knows, all the same, but that some day variations in opsonic index and in the powers and shapes of the blood-cells may be found as they arise in bed or at work, on a full or a low diet, and that some day it may be possible to measure these variations with scientific accuracy and to utilise them in strict accord with the effect we wish to produce? The day assuredly is not yet, but I have no doubt that Shakespeare's remark, "there are more things in Heaven and Earth than are dreamt of in our philosophy," will be found to be of transcendental importance here as elsewhere in the realms of natural science.

Feeling so strongly, as I do, the immense importance of my subject, it is nevertheless somewhat difficult to know how to handle it in the way best calculated to arouse your interest, to sustain your attention and to leave you with some crumbs of practical information for future guidance, and yet at the same time not to weary you with useless repetition.

Upon diet in acute disease I have recently spoken, and my words have been published in the CLINICAL JOURNAL, in our Hospital gazette, and as a reprint for private circulation, so that I shall say nothing more to-day upon that half of my subject.

Bed or rest, the other half of my subject, by a very natural expansion of ideas, includes also sleep, and to these points I shall devote the rest of my time, and I propose to consider them first from a general point of view, and later to follow the application of these principles to a few special diseases.

Physiology teaches us that when asleep katabolism is at its lowest ebb in the body, that the main functions of respiration and circulation are carried on purely in a reflex manner, in strict relationship to the needs of the tissues for a due supply of oxygen and other nutrient elements by means of which they may perform their anabolic functions. We need not, however, go to physiology for a proof of the statement, for in every baby we have a highly practical demonstration of the fact, a mathematical proof of the truth of the assertion, and every invalid will tell you of the marvellous effects of an hour or two's quiet sleep.

"Mental repose, bodily comfort, moderate warmth, a certain amount of fatigue combined with perfect quietude are the physical conditions or essentials for the production of sleep"—a quotation from a lay paper which very excellently sums up the position, and it is quite germane to my thesis to consider how we can best provide these essentials for a patient.

The moderate warmth should be easily obtainable, and the perfect quietude in the sick room is a matter upon which we can instruct a nurse or mother to insist; a substitute for the certain amount of fatigue is often supplied by the disease in the shape of excess of waste products in really acute illness, but in more chronic troubles and in convalescents is easily obtained by allowing a degree of exercise suitable to the occasion—a point we shall have to return to later; as for mental repose and bodily comfort they are factors of immense importance in the management of disease, and come under the definition of the treatment of symptoms as they arise, for "symptom of disease" is essentially "a lack of mental repose and bodily comfort" (the late Dr. Sutton used to commence his lectures with spelling disease dis-ease).

To discuss this treatment of symptoms adequately is, of course, too wide a field for a single lecture, but it is not out of place to offer you a few general hints on the use of anodynes and hypnotics. A first general hint cannot, I think, be emphasised too much, which is this: Never allow a patient to know the name of the drug that he takes for the purpose of procuring sleep; it is by telling patients what they are taking that we directly encourage the appalling habit of self-drugging that now threatens the very foundations of social life.

The next general hint is to use a drug with the

whole action of which you are tolerably familiar; I am not in a position to hold the scales between veronal, trional, tetronal, sulphonal, antifebrin, antipyrin, morphia, and chloral, but I have just this to say, that so far as I can judge there is no member of the coal-tar series which has not a dangerously depressing effect upon the heart in certain people, and I am personally, after considerable experience, very chary of going beyond opium and its derivatives or chloral for the purpose of procuring sleep by drugs, though I very much prefer to give a trial to simpler measures, such as hot or tepid or cold sponging, or bathing the forehead with eau-de-Cologne, methods which are absolutely without danger. I would like to add that in doses limited by, say, $\frac{1}{3}$ gr. in the case of morphia and 25 gr. in the case of chloral, these drugs are NOT depressants of the heart and may be safely used.

The last point I wish to touch upon in the matter of procuring sleep is one that I should perhaps have put first, and it is this: before resorting to artificial means at all ask yourselves the question, Is the sleep worth the price that has to be paid for it? If the insomnia be genuine it may be that an hour's sleep is worth any price, but often, with chronic invalids especially, a lack of sleep exists only in the patient's own mind, and if you have any suspicion of this being the case make inquiries before taking further steps.

Sleep is the most complete form of rest, and I have thus dealt briefly with it first, but the primary object I had in view was an estimation of the value of the less complete forms of rest in disease and a consideration of the question, "When should we allow a patient up and advise him to get about?" And to this we will now turn.

Rest in this sense essentially means an absence of voluntary action of the muscles. Now it is from the action of our muscles that we obtain the great bulk of our bodily heat, and it is from the same source that a considerable portion of our waste products are derived, products that have to be eliminated through the skin, kidneys and lungs, and lastly, it is when our muscles are in voluntary use that our circulation and respiration are most actively at work; hence we can see at once a very direct connection between rest and diseases of skin, kidney, heart and lungs, and clinical experience teaches us very forcibly that there is an equally important, though perhaps less direct, and certainly more obscure in essential details, relation between rest and our other organs and tissues, the best illustration of which, perhaps, occurs in diseases of the stomach and intestines.

Now, in general terms, what does getting up mean? It means the use of muscles of voluntary movement, which at once increases the quantity of heat in the body and throws a greater amount of waste products into the blood; it means the quickening of the respiration and also of the circulation, and centuries of experience prove without any intricate physiological experiments and measurements that these three things all work in harmony to keep the body, its organs and tissues in the very best circumstances for health. It means also a change of atmosphere for the skin and generally for the lungs—two points which we are rather apt to overlook when considering the propriety of ordering a patient to get up.

Taking, then, a broad general view of "getting up," we see an underlying principle, viz. that provided the organs are healthy (or recovering from a diseased condition) whose work is increased we should not keep a patient from getting up if, and when, he feels inclined to do so. These organs are the heart, the lungs, the kidneys and the skin, and I propose now to consider disease of these four organs so far as it supplies an answer to the question, Can I safely allow this patient to get up and walk about?

Heart Disease.

As the heart is the most essential organ to life, and as its beat is (in the ordinary sense) absolutely continuous from long before birth to the last minute of life, it is but natural to assume that it offers us the most typical example of the difficulties of our problem; we cannot stop its work entirely—by what shall we judge that we ought to diminish or increase it?

We may, I think, usually divide its diseases into those of the nerves and those of the muscle, for though I believe that intrinsically 99 per cent. of deaths through the heart are due to myocardial weakness or incompetency, there are a few exceptional troubles, such as cerebral tumours and other coarse brain troubles, or perhaps diphtheritic or alcoholic neuritis of the vagus and other rarer nerve diseases, in which the fatal event is brought about

by, or through, the cardiac nerves, but valvular disease I think by itself, *i.e.* with absolutely healthy muscle, is practically never the cause of death.

In this connection there is a useful hint I can give you from experience: symptoms of heart disease are of two kinds—(1) local consciousness of possessing a heart, (2) distant symptoms of imperfect circulation through organs. If the latter are present, either alone or in combination with the former, the case is assuredly one of myocardial insufficiency; if the former are alone present the case is far more likely to be one of reflex disturbance of the heart through its nerves, and if in addition there are no bruits and the first sound is well differentiated from the second, then this likelihood becomes almost a certainty; the importance of the point lies in this fact, that for these nerve disturbances we need not be very anxious about giving the heart ordinary work to do, and may safely encourage the patient to fulfil his ordinary duties, provided, of course, we treat such common reflexes as indigestion, etc., with which I am not now dealing; in other words, rest in bed or invalid life about the house is not indicated in such cases. A very typical case of this sort occurred to me a little time ago: A woman, about thirty years of age, suddenly felt faint in church. She went home; a doctor was sent for, who ordered her to bed for a few days, which lengthened themselves to a year before I saw her. I could find no evidence of real heart disease, but took her into hospital for observation, and within a week had her about the ward helping with all sorts of work. I sent her to a convalescent home, where she also helped in the work, and she returned home. Within a few months she again found bed an irresistible attraction, and I happen to know she has remained there for the last five or six years in daily expectation of death according to her own account—a burden to her husband in painful reality, a fraud and sham, a typical example of the evils of suggestion on a weak-willed woman.

Exophthalmic goitre with severe heart symptoms is an exception to this rule I admit, but hardly comes within our present purview and needs other and fuller consideration, for it is probable that this disease does effectually and permanently debar its victim from more than the very lightest of domestic duties.

Turning now to the commoner forms of organic heart disease, mainly valvular but also myocardial, if the patient complains of any symptoms at all it is quite obvious that the work he has given his heart has been greater than the organ could stand with any degree of comfort, and equally obviously the simplest plan is to reduce that work by rest in bed, and another important point is to clear out the primæ viæ, to try to break the vicious cycle of stomach irritating heart and heart retaliating on the stomach.

So much is common routine, and drugs, too, will be administered and the patient will feel better or worse, and in the latter case various combinations of drugs will be tried, with which I am not now dealing, but it has to be remembered that the heart's work is still on a plane below that of ordinary active life, and the point I wish to deal with is, How can we tell when or whether the heart is capable of rising to the necessary level of energy?

Inasmuch as a very large proportion of such cases are due to fresh slight attacks of endocarditis, the first point is to determine that anything in the nature of acute inflammation has subsided, for here acute inflammation means softening and swelling of the valve, and extra work thrown on to a soft valve means in all probability increased deformity of the valve *qua* its function, and, therefore, in all probability permanent increase of the effect on the blood-stream—either regurgitation or obstruction.

The best indication that inflammatory processes have ceased is a normal temperature, and to determine that it is normal it should be taken four-hourly, and my own plan is to have the temperature normal for three weeks at least before allowing the patient to rise from bed, and even then to prop him up in bed for a few days as a preliminary trial. I then put into force the following scheme: count the pulse frequency while the patient is lying, count it again on sitting up in bed, and then on standing, and then on taking a step or two. Now, normally between lying and sitting up, between sitting up and standing, and between standing and walking there should be an average difference of, say, five or seven beats a minute, and my rule is this: if in a given patient the difference amounts to much over ten beats a minute progressively for each increase in work I consider further rest is indicated, or, at any rate, that we must proceed with very great caution, and only on the urgent wish of the patient allow him to be placed on a sofa or couch. A further detail is to reverse the process and note the time taken by the heart to resume the rate of beating of the greater degree of relief from work, this reverse process being a fairly accurate clinical indication of the health of the myocardium.

While speaking of this method of determining the condition of the heart I may add that it has an application perhaps of even greater value in those cases in middle and later life, or even in young

subjects, when either the patient complains of his heart, or when, from the nature of his symptoms, shortness of breath, etc., we are led to suspect that the heart is not sound, but cannot hear any corroborative bruits. In these cases, *vide* my lecture in the CLINICAL JOURNAL, "Morbus Cordis *sine* Bruit," I add to the above counts of the pulse one made after the patient has hurried a good deal, and as above note carefully the results, and especially the time taken to regain the frequency it had previous to the periods of greater exertion. I consider this the most valuable clue to the condition of the heart, and one upon which I almost entirely rely.

Suppose now that the heart's response to this test be satisfactory, or fairly so, there can be no doubt about the propriety of allowing the patient to get up and try to gradually resume his normal life with pauses determined only by the fact that my clinical rule of frequency is being flagrantly broken. There will then arise the further question, Can I push matters a little further, and can I by means of graduated exercise get the heart into a better condition than it was at the commencement of the trouble which brought the patient to me?

Theoretically we know that exercise strengthens muscles and improves their general health, and for twenty years or more I have steadfastly advocated carefully regulated increase of work for those with hearts not too hopelessly crippled; later the method was taken up by interested people and boomed in and out of season, and became fashionable in connection with the name of a medical man, Oertel, and with the name of a place, Nauheim, and its baths; fashion spoils many good things, and leads to unsuitable cases being treated at home and elsewhere without due care and supervision; the fault lies, not with the method, but with the advertisement of it.

Just one other point, gentlemen, and I have finished with the general management of heart troubles. Mental repose we have stated is a great element in sleep and in maintaining health; now a very great help to mental repose is an impression of improvement made upon the patient himself, and this impression is almost invariably produced on a patient by permission to get up. His mental attitude is this: "My doctor is doing his best for me; he thought I was so ill that I must go to bed, he now thinks I am much better or he would not let me up, and, by Jove, I do feel better; I'll get up." Don't despise such homely feelings, gentlemen; they inspire hope, and where hope is there is a chance, and when hope departs from our patients I might say our functions as healers are practically useless, and only those of the parson and the undertaker are of much avail. Remember this in all diseases, though mentioned only in connection with the heart.

December 7th, 1908.

DISEASES OF MINERS, WOOL-SORTERS, AND OTHERS.*

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

IN my last lecture I was speaking of the disastrous effects of dust, and specified some of the many industries where the workman is exposed to this danger. I told you how the lungs of the men employed in steel grinding and other dusty work become impregnated with dust, how the lungs are slowly turned into a hard, almost solid material, which quite unfits them for the function of aëration. Either the patient succumbs to lung trouble directly due to this, the lung being incapable of getting rid of the dust that slowly stifles it, or tubercle bacilli, entering the lung, find there a suitable soil for development. I will now speak of some further industries where there is danger of lung disease consequent upon dust.

Some years ago consumption among coal miners was very prevalent in the coal mines of Great Britain, but in no occupation have the good results of legislation been more marked than in this.

Coal mines are now well ventilated, and consequently the cases of consumption have remarkably diminished. In fact, so free from this disease are miners, compared with other working men, that some authorities believe coal dust to be antiseptic and actually a preventive of consumption. Whether this is really the case is doubtful, but the good air now pumped into the mines, better housing and drinking water, good wages, increased temperance, the establishment of reading and recreation rooms, and the consequent improved physical and moral condition of the miners, are enough to account for the good results.

The same satisfactory account reaches us of the condition of coal miners in Germany, Belgium, and France.

There are few diseases to which the coal miner is specially liable, and it may, on the whole, be considered a healthy occupation, but although I have told you that he is specially free from consumption, there are certain non-tubercular lung

* A Lecture delivered at Gresham College, adapted from 'Diseases of Occupation,' by Sir Thomas Oliver.

diseases caused undoubtedly by the inhalation of coal dust.

Fortunes have been made in an incredibly short time in the *gold mines* of South Africa by young English miners, but many of these men have returned wealthy to their native village, four or five years after leaving it, then full of strength and enterprise, now broken in health and far advanced in consumption.

Sir T. Oliver was the first to draw the attention of the Government and the mining officials to the high death-rate from chest troubles among the South African gold miners, and Mr. Chamberlain then appointed a commission to report upon the subject. A very bad state of affairs was revealed. The race for wealth has alike blinded employers and employed to the lives that are at stake, and sufficient care to guard against accidents and disease has most certainly not been taken. I will speak now only of the risk from dust to which these men are exposed. The danger lies not in the coarse particles that are chipped off the rock, but in the fine, impalpable dust drawn into the miner's lungs when he is breathing deeply at his heavy work. After death the lung is seen to be streaked with long, dark lines of pigment, marking the course by which dust particles have been carried. The disease of the lung thus brought on is called "miner's phthisis," which is not due to tubercle, but directly to the effect of the dust the patient has been breathing. The men who work the *rock drills* are more especially the victims of this illness. Rock drills driven by compressed air raise a larger quantity of dust than do hand drills. The blasting, drilling, shovelling and breaking of the rock produce much dust, and under the microscope this dust is seen to consist of sharp particles of flint. The average age at death of rock drillers is under thirty-five years, after seven to nine years of this work. It is strange to think how readily men will run such risks for the sake of gain. They are themselves often to blame. In their hurry to attain wealth they rush back into a mine too soon after blasting operations, knowing well what the dangers are, but allowing neither the gas to escape nor the dust to settle; they do not give themselves time for washing before food, and they work too many hours each day. As they can earn as much as £50 or £60 a month they hasten to be rich and forget the risk to their lives.

The condition among the Kaffir labourers is equally bad, and sufficient care is not taken for their welfare. The state of the Chinese is better; they are themselves more intelligent and healthier, but it is to be feared that many of them will carry the seeds of consumption back into their own homes—consumption contracted in South African mines.

Of recent years mine owners have introduced improvements, and if they will continue their efforts to remedy the hygienic condition, the bad reputation these mines now suffer from will soon improve.

Wherever the rock drill is used there should be a spray of water kept playing in and around the hole to lay the dust, and this applies to all mines where this machine is employed.

In the *quartz mines* of Bendigo in Victoria the cases of consumption have more than doubled since the introduction of the rock drill. Here the deaths from accidents have decreased, whilst the deaths from tuberculosis have alarmingly increased, the death-rate among the miners being six times greater than among other men.

This calls for improvement in the hygiene of the mines, for we must consider, not only the misery it brings to the unfortunate miner himself, but how the infection is carried into his home and to his belongings. As a matter of fact it has been found that the wives of the Bendigo miners die in larger numbers from consumption than other women in their neighbourhood. The common enemy is the dust of the mines.

Even worse is the condition of the ganister miners. Ganister is a very hard stone found in Yorkshire, Durham and Derbyshire, and is wanted to make a special kind of brick which is used in iron works. The men employed in these mines suffer so much from consumption that it is wonderful that any persons should be found to take up the work. In the drilling of the rock for blasting and after the explosion, by which process alone this stone can be moved, the air is filled with a fine dust which the miner must necessarily inhale. After the stone has been brought to the surface it is ground in powerful mills, and the men employed in this process, the ganister grinders, are those who suffer most. The mortality of the grinders is eight times as great as among bricklayers and four times as great as among the miners.

The death-rate per thousand is still 179, in spite of steam jets and sprinkling to lay the dust.

Tin mining has long been considered a dangerous trade, and the cases of lung disease among the miners are numerous. Here, again, the men who work the rock drills are the worst sufferers, and we must conclude that the dust thus raised is the cause. The lungs are weakened by irritation from dust, the tubercle bacillus enters and converts the case into one of consumption.

The health of the men working in slate quarries and the consideration of their labour was the subject of a report by a Departmental Committee in 1895. It was found that this form of mining was not free from accidents, owing chiefly to falls of earth from the roof and sides during the process of getting the slate. But although phthisis does occur among those men who work underground, the cases are not so frequent as among the workers in the slate mills, where a great deal of dust is inhaled. On the other hand, there are more cases of pneumonia among the miners, probably due to chills and exposure to damp and cold when the men come out of the warm mine into the open air. Their resistance is lessened by fatigue after the day's work, and they fall easy victims to the dangers of a sudden drop in the temperature.

Dr. Hamaide, a French doctor who has made a special study of slate-miner's diseases, tells us that he has seen men suffering from slate-miner's phthisis spit up fragments of a hard stony material, accompanied by profuse bleeding from the lungs. Dr. Arlidge reports the same kind of thing happening among the lung-affected potters of Staffordshire. In most of these cases of phthisis no tubercle bacilli are found in the expectoration of the patients, which points to the fact that the origin of the disease is the dust inhaled.

We are never tired of insisting upon the fact that an open-air life and open-air occupations will make a man less liable to contract consumption, or, having it, will often lead to his recovery. But there are exceptions to every rule, and when we have the enemy dust to contend with even an entirely open-air occupation will not always save a man. Stonemasons and marble cutters are the sad exception to which I now allude.

The death-rate among these men from consumption is six times higher than among city clerks, whose work is indoors. But from what I have told you, you will easily see the reason. These masons inhale the sharp, hard dust from the stones they are chipping, the disease in their lungs develops slowly from the constant irritation of the lung tissue, and, as in so many other cases of dust poisoning, the tubercle bacillus ends the chapter.

Mill-stone builders suffer in the same way. They work in the open air, but they inhale dust from the very hard stone they are dressing and from the steel of their tools. Their wages are high, as much as 50*s.* or 60*s.* a week, but the men are intemperate and the risks of consumption great, and few of the men live more than ten or fifteen years in the trade. Some few of the more careful men use sprays to water the stone they are chiselling, with the result that with the laying of the dust their health is better.

In a popular lecture delivered at the British Medical Association Meeting at Sheffield by Mr. Edmund Owen, he tells the following anecdote:

"Before a medical society in London were brought parts of the lungs of a mill-stone dresser. For twenty years this man had been worried by a morning cough, but he never had to give up work for it. Then one cold night he caught a chill, after which he quickly lost flesh and strength and he looked as if he were in a consumption. But he was not consumptive; the doctor said his disease, which in due course proved fatal, was due merely to the constant worrying of the lungs by the gritty particles which he had taken in with his breath whilst at his dusty work."

In a previous lecture I told you of the many cases of lead poisoning that occur among the manufacturers of *pottery* owing to the lead employed in the glazing, but the danger from lung troubles is another risk to be encountered in this industry, and it is one of the trades most affected by this scourge.

"Potter's rot" or "potter's asthma" has long been recognised as an evil prevalent among them, and the two dangers of chest troubles and lead poisoning have always caused pottery manufacture to be placed high among the dangerous trades. Thanks to improved methods of working and to the introduction of fans and strong draughts to carry away dust from the face of the workers their condition is improving. The air of potteries has been analysed and found to contain particles of earth, granite, flint, dried glaze from the ware, soot and

wood charcoal. This is the explanation of the chest troubles so many pottery workers suffer from. The dust makes a breach in the epithelial lining of the lung, reduces the resistance to micro-organisms, and thereby paves the way for the entrance of the tubercle bacillus.

In the lungs of people working at dusty occupations corroboration of their employment is found after death, proving, if proof be needed, that the dust they have persistently inhaled has wrought the mischief and has led to their destruction.

I could enumerate many more trades where the workers are exposed to dust and where they suffer in consequence, but enough has been said to show you what the danger is, and how we should make every effort in our power to lessen it. I will now pass on to describe to you two diseases of occupation caused by parasites.

The first of these is a disease to which miners and tunnel makers are specially subject in Northern climates. I have myself had a large experience of it in Egypt among the peasants, who lead a very different life in their sun-lit fields to the underground workers we are now interested in, but who have some bad habits in common with miners. I am speaking of ankylostomiasis, or hook-worm disease.

During the tunnelling of the St. Gothard the workmen were attacked in alarming numbers by anæmia, shortness of breath and swollen feet. The illness, which was then not diagnosed, was thought to be due to the bad feeding of the miners, to chill and to the lack of proper ventilation in the tunnel.

But Perroncito, of Turin, discovered in the intestines of some of the men who died large numbers of small, white, thread-like worms, and it was then recognised that the symptoms were due to ankylostomiasis, a disease known in hot climates among workers in earth. It is possible for one patient to infect an entire mine, for the larvæ of the worm, when deposited on the damp earth, survive a long time, and gain admission into the human body through food, the unwashed hands of the workmen being soiled with earth containing larvæ. Another cause of infection was lately discovered by Professor Looss, of Cairo, who has made a most minute study of this worm, and who found that he had accidentally infected himself during his experiments, the larvæ having penetrated the skin of his hand and thus entered his body, travelling along the veins, through the heart, the capillaries of the lung, alveoli, bronchi, trachea, larynx, œsophagus, stomach and duodenum, where they become sexually mature.

The worm, which is about one third of an inch in length, attaches itself to the mucous membrane of the intestine and sucks up nourishment. In doing so it deposits in the bitten surface of the mucous membrane a poisonous material, which causes the symptoms in the patient. These symptoms are slowly developed, at first causing weakness, indigestion, pain and great pallor. The feet become swollen, the pulse weak, and if the patient is re-infected, as he often is in an infected mine, the symptoms all become worse and he may die of fatty degeneration of the heart and kidneys.

The tunnel makers of the St. Gothard, having completed their work, were distributed all over the Continent to other tunnels or to mines, carrying everywhere the infection with them. In this way the disease is spread. For instance, in 1840 the coal fields in Westphalia attracted no less than twenty thousand miners from Posen, Poland, Hungary and Italy, and with them came ankylostomiasis, brought by one or more of these immigrants, and the mines have never since been free from it. In 1902 there were 1355 cases in the mines of one valley alone, and in three years it has cost the Miners' Union no less than £100,000 to grapple with this disease. Too high praise cannot be given to Germany for the business-like and thorough manner in which this trouble has been dealt with, and success will to a great extent follow these endeavours.

Some alarm was caused a little time ago when the disease was discovered in the tin mines in Cornwall, where the men had been known for some time to suffer from debility, anæmia, and the other symptoms of ankylostomiasis, but, speaking generally, English mines are comparatively free. All new comers, especially those coming from hot or infected countries, should be medically examined before entering a non-infected mine, the hygiene of the mine should be good, ventilation well attended to, sanitary conveniences should be placed in every mine, and the miners themselves should be instructed in the life-history of the worm and the dangers of want of care and cleanliness. The disinfecting of infected mines

has been found extremely difficult, as the larvæ are very resistant to all disinfecting solutions, but it has lately been found that a strong mixture of common salt and water destroys the larvæ easily and inexpensively, and it is to be hoped that this means will be found effectual in destroying the worm where it has already found a home in mines.

The skin entry of the larvæ of this parasite has been thoroughly worked out on dogs by Professor Looss, and has been confirmed by Schaudinn in Germany, Lambinet in Belgium, and Claude Smith in America.

From time to time anthrax, or wool-sorter's disease, has been a veritable scourge in Bradford and in the woollen districts of Yorkshire, whilst London and Liverpool have not been entirely free from it. During the six years 1899 to 1904, 261 cases were reported to the Home Office.

This terrible disease is due to a minute organism in the blood of animals. The danger to human beings lies in the handling of hides or fleeces of animals which have died from it. The most minute scratch on the hand is sufficient to enable the bacillus to enter the body, or it may be inhaled with the dust from the hides, or swallowed with food. It is always blood-stained wool or hair that is the carrier of the germ of anthrax, and dust is dangerous because it contains particles of dried blood.

Although about 1000 cases of anthrax occur in animals in Great Britain every year; it has never been the scourge here that it has been in Germany, where at least four times as many cases are recorded, whilst in European Russia and the Caucasus there are about 40,000 cases every year. It is from Asia that most of our infected material comes, and in consequence of the prevalence of the disease abroad those factories that import most foreign wool have suffered chiefly. In the woollen districts of the South of England and of Scotland anthrax has been conspicuously absent, owing to the fact that only colonial and home grown wool is used there.

We find most cases among wool-sorters, wool-combers and spinners, and among those who manipulate horse-hair for stuffing chairs and mattresses and bristles for brushes. Farmers, tanners, butchers and meat inspectors have also been the victims of it, besides those who have been exposed to the inhalation of anthrax spores in dust arising from the products of dead animals.

The anthrax spores by means of earthworms (as discovered by Pasteur) can contaminate a field where infected carcases have been buried. Thus in 1901 two horses ate grass off a field in Yorkshire close to a wool factory, upon which dust from Persian wool had been blown, and both animals became infected. Again, in Pennsylvania sixty cattle died of anthrax after grazing in a meadow watered by a stream contaminated by tannery refuse.

Undoubtedly the greatest danger to people is the dust from infected wool, but where wet hides are used, or where exhaust ventilation has drawn the dust away from the faces of the workers the results have been good.

All wool coming from infected countries should be thoroughly disinfected, as is now done by law in Germany, where anthrax has, in consequence, much decreased of late.

There has been, of late years, considerable legislation in this country to protect the factory hands, and disinfection is insisted upon, but this disinfection of suspected material must be efficient to be of any use, and not every disinfectant will suffice.

Horse-hair, by order of the Home Secretary, is shortly to be compulsorily disinfected by importers before distribution, and it is to be hoped that bristles will be similarly treated.

The effects of anthrax poisoning differ in individuals and according to the mode of infection. Sometimes a hard, red swelling appears, usually on the face or neck, which must be cut out by the surgeon. If this is not promptly done it causes an enormous inflamed swelling all round the pustule, the temperature rises, and the patient in all probability dies. Here, for instance, is a case of Dr. Eurich's from Bradford:

"The illness began with a small pimple on the man's wrist on February 25th, 1907, but it was considered so trifling that medical advice was not sought till February 27th. The bacilli of anthrax were then found in the serum of the pustule. An hour or two afterwards the pustule was excised, and three ounces of Sclavo's serum injected during the course of the next forty-eight hours. The patient never responded to the treatment, but became delirious and violent, and died on March

1st in a state of coma. At the autopsy numerous hæmorrhages were found in the lungs and extensive hæmorrhages on the brain."*

Wool-sorter's disease is a distinct form of anthrax which attacks the lungs, and which is caused by inhaling the dust from infected wool. Although it is extremely fatal there are no very acute or painful symptoms, but the patient soon shows signs of collapse and usually dies on the third or fourth day.

Up to a short time ago the treatment of this pulmonary anthrax gave very unsatisfactory results, but Dr. Sclavo, of Milan, has given us an anti-anthrax serum, which is injected into men or animals, with excellent results. The peasants in Italy have so much faith in it that they insist on having this rather than any other treatment.

Precautionary regulations have been adopted by the Home Office, insisting upon disinfection of hides, removal of dust by means of fans and hygienic care of the workers. Excellent results have followed the enforcement of these rules.

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SOME POINTS IN THE DIAGNOSIS OF MALIGNANT DISEASE OF THE LIVER.†

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It would be impossible to deal adequately with this large subject within the limits of the short time at my disposal, and I shall refer only to certain points in the diagnosis of cases presenting themselves as possible malignant disease, whether primary or secondary, of the liver substance. The diagnosis of malignant disease of the gall-bladder and larger bile-ducts, except in so far as they are combined with malignant disease of the liver, will not be considered here. In these remarks no references will be made to those cases of malignant disease of the liver in which enlargement of the organ is not manifest during life.

The question whether in a given case there is malignant disease or one of the other less serious causes of enlargement of the liver is often one of the easiest to decide, but in some of the cases when seen for the first time in the stage of comparatively slight enlargement the decision must be left in suspense in order to watch the progress of the case and the effect of treatment. Although evidence of the existence of malignant disease elsewhere in the body does not make it absolutely certain that hepatic enlargement is of the same nature, the probabilities that it is are so overwhelming that these cases need not be further considered. The presence of glandular enlargement above the left clavicle is a most valuable confirmation of malignant disease inside the abdomen, but its absence does not seriously militate against the existence of malignant disease of the liver when this appears probable for other reasons. I have looked for it in a large number of cases and in the great majority have not found it.

A means of diagnosis which only requires very brief mention here is the simple course of opening the abdomen and looking at the liver. This should not be undertaken—at least with this object—unless there is a fair probability of finding a condition that is remediable, such as gall-stones in the common duct. When the abdomen is opened and inoperable malignant disease found, the patient is in a worse condition than before—partly from the effect of the operation, and partly because the knowledge of the failure to obtain relief and of the real nature of his disease is likely to dawn on him.

After referring in general terms to the diagnosis of malignant disease of the liver from the conditions which most closely simulate it, namely, an enlarged cirrhotic liver, hepatic syphilis, hydatid, I shall mention examples of a few rarer causes of enlargement.

A large cirrhotic liver is extremely likely to be confused with malignant disease in a comparatively early stage, but only at this period, for cirrhosis never produces the extreme enlargement seen in advanced cases of malignant disease. Extreme enlargement, extending below the iliac crest, is, provided a movable or displaced liver or a tongue-shaped lobe is eliminated, almost certainly malignant. Although the presence of definite nodules and of umbilication—the latter being far from common and subject to some fallacies—is very strong evidence of malignant disease, the absence

* 'Diseases of Occupation,' p. 342.

† Delivered before the Medical Society of London.

of these signs does not put this diagnosis out of court, for there may be a single large primary growth embedded in the substance of the organ and expanding it, or there may be a diffuse infiltration with secondary growth. Uniform enlargement is rather in favour of cirrhosis; enlargement confined to the left lobe—though not common—of new growth. The association of a palpable spleen is a valuable sign in favour of cirrhosis, and although I have seen it in cases of malignant disease, it is unusual and due to some complication such as thrombosis of the splenic vein. The combination of ascites and jaundice is seen in both cirrhosis and malignant disease of the liver, but is commoner in growth. In a patient with the abdomen distended with ascites it is often impossible to express any opinion until, as a result of tapping, some impression can be formed as to the size of the liver. In cirrhosis jaundice is less prominent, and may be transient.

Cyto-diagnosis of the ascitic fluid withdrawn from a case of doubtful cirrhosis or malignant disease of the liver can hardly be relied on. In cirrhosis the effusion is passive and the predominating cells are endothelial cells, but malignant disease may produce a mechanical ascites by pressure on the portal vein or its branches and so imitate the ascites of cirrhosis. Conversely in a woman who really had cirrhosis the cytological report, from the presence of multinuclear endothelial cells and endothelial cells showing mitosis, suggested intra-abdominal growth. In malignant disease the onset of jaundice, though usually gradual, painless, and progressive, is not so in all cases. It may come on with the symptoms usually considered to be characteristic of "catarrhal jaundice," namely vomiting and diarrhœa. With such an onset the jaundice may steadily persist and deepen, or it may lessen or even intermit in such cases. It is probable that in the first instance slight pressure on the common or other large duct so diminishes the resistance of the duct that a mild grade of infective cholangitis is set up; if the pressure on the duct does not increase, the biliary obstruction diminishes as the cholangitis subsides; if, on the other hand, the pressure on the duct progresses, absolute obstructive jaundice may result.

The history of alcoholism and the age of the patient have some bearing on the diagnosis. A considerably enlarged liver in a patient over sixty years of age, especially if not obviously alcoholic, is more likely to be malignant than cirrhotic, especially if the patient has previously been free from all minor ailments. Too much importance, however, must not be laid on the history. I have known several instances in which malignant enlargement of the liver in individuals connected with the liquor traffic was regarded as cirrhotic, partly, no doubt, from the humanitarian motives of avoiding the fatal diagnosis, but partly from the association of ideas.

Variation in size is important; a malignant liver increases in size, though its rate of progress may be irregular; a cirrhotic liver may certainly show some increase in size, for example after a drinking bout or after intercurrent influenza, but on the other hand, it may diminish in size. Although from increased abdominal distension a malignant liver may be less easily mapped out, no real diminution ever occurs.

Hepatic syphilis, especially gummata on a lardaceous liver, may closely imitate malignant disease. In such cases the history of syphilis is not enough to guide us, as malignant disease may of course supervene in a syphilitic subject. The most trustworthy guide is the effect of a thorough anti-syphilitic course of treatment.

Concomitant splenic enlargement, albuminuria, and diarrhœa are important as pointing to lardaceous changes in the spleen, kidneys and intestines; these conditions are very rare in malignant disease of the liver.

The nodular and deformed liver due to gummata and cicatrices may also imitate malignant disease, and in this connection it is interesting to remember that before the true nature of the visceral lesions of syphilis was recognised gummata were regarded as cured malignant disease. The gummatous liver does not increase in size in the same manner that a malignant liver does, and though it may give rise to pain and ascites there is seldom the same degree of constitutional disturbance as is seen in malignant disease.

Inherited syphilis may in certain instances imitate sarcoma of the liver. Marchand described cases in infants of tumour growth of the liver ascribed to this cause, and Morley Fletcher recorded a case in this country in a baby, æt. 7 weeks. Neither in Marchand's or Fletcher's cases was there a satisfactory history of syphilis, the

diagnosis depending on the histological appearances.

Last summer I saw a boy, æt. 9 years, a Mongol, who had improved under thyroid treatment, but had developed a temperature varying up to 102° F. at night and a large hard liver. I regarded it as malignant and advised against operation; others saw the patient, and eventually, although a blood-count was made, it was thought that the abdomen should be opened to eliminate suppuration. The appearances at the operation were described as like new growth or of some unclassified type of growth of unusual rarity. Various opinions as to the nature of tissue removed at the operation were expressed; fibro-sarcoma, inflammatory, or syphilitic. Personally, from examinations of the slides I have no knowledge that it was syphilitic. Three months after the laparotomy the boy was very well. In this family the parents were healthy and with the other children did not show any sign of syphilis. It was, indeed, suggested that it might be a manifestation of syphilis in the third generation.

From hydatid of the liver.—As far as the physical signs go there may be a very close resemblance between a large primary malignant growth expanding the liver and a hydatid cyst in the same position. It is then mainly by the consideration of other factors, such as the patient's condition and history, that the diagnosis is made. In hydatid the growth increases very slowly and is painless, and does not affect the patient's health and strength unless it suppurates.

In malignant disease the growth increases comparatively rapidly in size, there is pain and usually tenderness, and the general health is affected sometimes even before enlargement of the liver can be detected. In other instances several distinct hydatid cysts may project from the surface of the liver and give rise to a knobby condition of the organ. Sometimes distinct fluctuation can be made out, or a definite hydatid thrill can be obtained. Multiple hydatid disease of the abdominal cavity may also imitate a large malignant liver. The margins of adjacent cysts which are in contact with the liver may be mistaken for the lower border of that organ.

In a large liver in a young person with soft projections, compatible with the view that they are cysts and with good general health, the question of hydatid arises. The presence of eosinophiles is in favour of hydatid disease, but its absence proves nothing. In the case of a large cyst an exploratory operation should be undertaken, as its presence is a distinct menace from the risk of traumatic rupture. Although no bad result invariably follows aspiration of a hydatid cyst, it is a risky procedure and should never be undertaken.

Besides hydatid disease almost the only chronic conditions likely, from the size of the liver they induce, to imitate advanced malignant disease are cystic disease and leukæmia. Cystic disease may give rise to great increase in the size of the liver. It is very chronic and is associated with similarly polycystic kidneys, with which, rather than with the liver, any symptoms present are correlated.

From the various forms of suppurative hepatitis.—It is important to realise that fever may occur in malignant disease of the liver. It is true that it may be absent entirely throughout the course of the disease, especially when, as sometimes occurs, the rate of growth is slow.

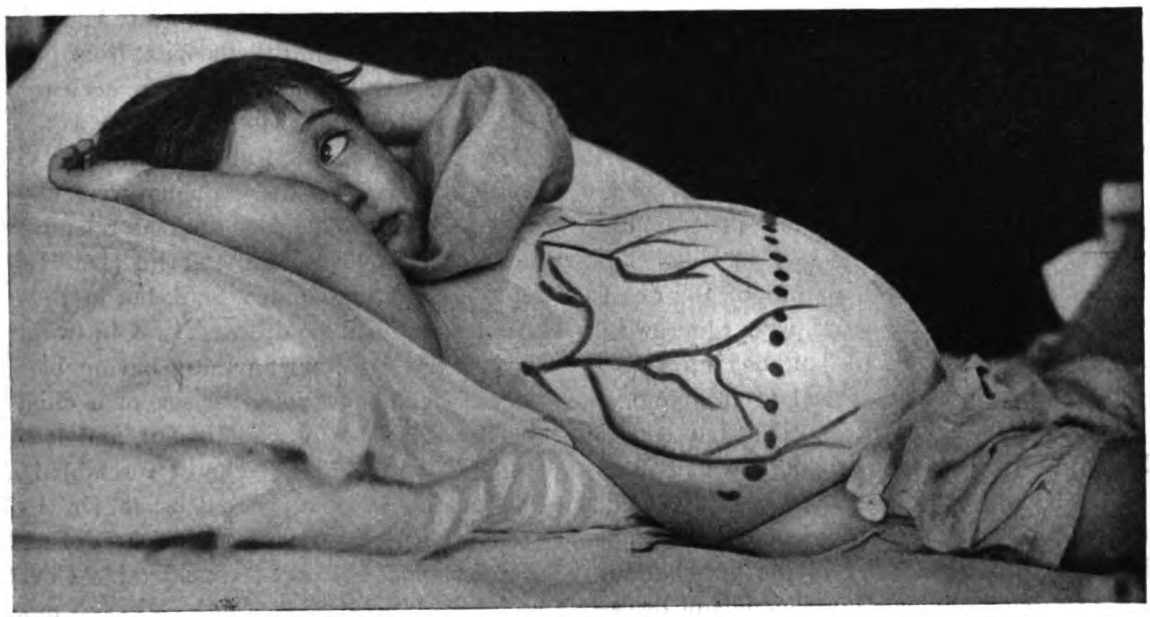
The occurrence of fever in cases of malignant diseases of the liver may be considered under two headings:

(1) In uncomplicated cases a certain amount of fever is not uncommon when the growth is rapidly increasing in size. In such cases the temperature may be raised for weeks, but it rarely goes above 102° F. This form of fever is specially apt to occur in sarcoma of the liver in young persons. It may be diagnosed as pylephlebitis, especially if there is any history of pain in the right iliac fossa.

(2) *Fever due to complications.*—The range of temperature is much wider than in the previous form, and resembles that of simple suppurative hepatitis. This may be due to microbic infection of the liver and suppuration in the immediate neighbourhood of growths or in the bile-ducts. For example, a carcinoma of the biliary papilla may give rise to secondary growths in the liver, and as a terminal event to suppurative cholangitis. Again, suppuration may occur around the primary growth in the colon or gall-bladder in a case with secondary growths in the liver. In rare instances the resemblance to simple hepatic abscess is rendered all the closer by a fluctuating area, palpable through the abdominal walls, due to a softened or hæmorrhagic growth, especially in cases of sarcoma.

A wandering liver rarely imitates malignant disease; its mobility and absence of the hardness and prominence seen in malignant diseases of the liver are important signs. Partial hepato-ptosis, the tongue-shaped or Riedel's lobe, is not very rarely diagnosed as malignant disease; and the condition may, if unrecognised, give rise to considerable difficulty when present in a patient, nearly always a woman, who is ill from other causes, such as grave anæmia.

Chronic venous engorgement of the liver should very rarely be taken for malignant disease of the liver. Hanot described such cases under the name of "hepatic asystole," in which, from the absence of any cardiac murmur and the size of the liver, attention was focussed on that organ. One such case, a girl, æt. 3½ years, was under my care in the Victoria Hospital for Children and was regarded as sarcoma. The liver progressively increased in size; there was ascites which required tapping on two occasions, some œdema of the feet, and the caval system of veins over the abdomen was very considerably enlarged (see photograph).



Photograph of a child with chronic venous engorgement of the liver imitating sarcoma. The dotted line shows the lower margin of the liver; the dilated veins have also been marked out in ink. (Photographed by Dr. Colin King.)

The heart was normal, but the pulse was rapid. There was nothing in the aspect or physical examination of the patient to suggest heart disease. The autopsy performed by Dr. J. Ledingham showed extreme venous engorgement of the liver. Here and there in the interacinar capillaries there were enormous numbers of streptococci, frequently forming embolic masses which entirely filled up the capillary lumen. The heart and pericardium, superior and inferior venæ cavæ and portal vein were all normal. It is difficult to understand the mechanism by which the extreme venous engorgement of the liver was brought about, inasmuch as the heart was healthy. I have elsewhere recorded the case of a woman with a large liver with referred pain over it, probably of anginal nature, as shown by the autopsy. Her appearance suggested morbus cordis, but the heart appeared to be normal. Her condition precluded much examination and she was kept under the influence of morphine. Three days before death she became jaundiced. At the autopsy the heart showed extensive fibroid change, the aorta presented syphilitic thickening obstructing the orifices of the coronary arteries, and the liver was enlarged, nutmeg, and free from growth.

Influenzal liver.—It appears that influenza may give rise to hepatitis. It is rare, but occasionally after influenza the liver becomes enlarged and there is fever. Of the form with a widely oscillatory temperature imitating suppuration it would be

out of place to say more here. But I have seen enlargement lasting a considerable time with an irregular and comparatively slight temperature, and malaise which very seriously aroused the suspicion of malignant disease in a man over sixty. In this instance I was inclined to the view that the influenzal poison stirred up a latent cirrhosis. The patient very slowly improved and was in fair health a year later.

Dr. DE HAVILLAND HALL asked whether the author had come across any cases of dysphagia as an indication of malignant disease of the liver, *i. e.* of a spasmodic nature as shown by the passage of a bougie. He had not seen such a case but had read of them. Possibly apparent stricture of the œsophagus might be of reflex origin as an outcome of malignant disease of the liver. Dr. Rolleston had not over-rated the difficulty of diagnosis in some cases of malignant disease of the liver. Even opening the abdomen was not always successful in arriving at a diagnosis. One case in which laparotomy failed to reveal the nature of the disease was that of a man, æt. 52 years, whom he saw in a nursing home; he had had jaundice upwards of six months, and lost a great deal of flesh and was much emaciated. The diagnosis rested between malignant disease and obstruction by gall-stones. Exploration was recommended to settle the diagnosis, and a leading surgeon opened the abdomen and came down on a mass in the portal fissure, which he at once concluded was malignant disease, said nothing more could be done, closed the wound, and he (Dr. Hall) told the man next morning that the mass could not be removed. The patient went home to the country and six months later he showed himself perfectly well. He had begun to improve soon after the operation, and he (Dr. Hall) concluded that the mass had been only inflammatory. That was ten years ago and the man was still in good health. In some cases even at the necropsy it was impossible to say whether a case was malignant or was hypertrophic cirrhosis without a microscopic examination. Gummata were sometimes very difficult to distinguish. He had recently under his care in hospital a patient with nodular enlargement of the liver with ascites. He did not improve under iodide of potassium and he got Mr. Spencer to explore. He opened the abdomen and found some masses which he felt certain were gummatous. The patient was put under brisk anti-syphilitic treatment, including inunction of mercury. Whether as a result of that or of the drainage at the operation he could not say, but the man made a good recovery, and that was eighteen months ago. He had seen the patient recently and he was in very good health. Primary hydatid of the liver might be difficult to distinguish from malignant disease of the liver. When he was house-physician at St. Bartholomew's a man was admitted with a semi-fluctuating tumour in the right hypochondriac region. While under observation for some weeks he gained five pounds in weight and improved in general condition, possibly because he had been admitted straight into the hospital from prison and had better food than he had been accustomed to. The tumour was regarded by Dr. Gee as abscess or hydatid of the liver. And as in those days exploratory operations in the abdomen were not readily undertaken, Dr. Gee suggested an opening should be made by potassa fusa, and he (Dr. Hall) went up to apply the blistering fluid, but as the patient seemed very bad he desisted, and in two or three days he died without anything having been done. At the necropsy a mass the size of a child's head was found in the liver, malignant, and it was soft and diffluent so that it almost resembled abscess or hydatid. He supported what Dr. Rolleston said with regard to fever in some cases of malignant disease. He had seen considerable elevation of temperature in such cases, though not more than 102° or 103° F. That might persist for some weeks, especially in rapidly progressing cases of malignant disease. He had been interested to hear about passive congestion of the liver, because the same difficulty had occurred to him. One case in particular, in which there was rapid enlargement of the liver and absence of any particular condition of heart which would lead one to suspect failure of compensation, the subsequent course of events and the result of the post-mortem examination showed that he was mistaken and that it was enlargement of the liver due to passive congestion.

Dr. SIDNEY PHILLIPS said the paper was so conclusive that little could be said in the way of addition or of criticism. He concluded that the author did not think symptoms were of much assistance in the diagnosis of cancer of the liver.

He (Dr. Phillips) agreed that there were no symptoms which would much assist in the diagnosis. Though Murchison used to include it among the painful enlargements of the liver it was often quite painless, and even when there was pain it was usually due to peri-hepatitis or to the growth of cancer elsewhere than in the liver. It was also well known that patients sometimes gained in weight when they had cancer of the liver, possibly due to the enlargement of the tumour itself. Jaundice and ascites were so seldom present, at any rate at the beginning of the disease, that they were not of any diagnostic import. Therefore, the diagnosis of cancer of the liver seemed to hinge on what was the nature of the enlargement. The enlargement of the liver presented itself under two aspects, one without jaundice or ascites and the other with. Of the enlargements of the liver without jaundice hepatitis was the one which most resembled cancer. Hydatid had been mentioned by Dr. Rolleston and by Dr. Hall. But this disease now appeared to have almost died out of London of late years, so that it was seldom one had to take that condition into consideration in making a diagnosis. But hepatitis was as common as ever, and the difficulties in diagnosing it from carcinoma were very great. With regard to nodules on the liver, even when present, they often could not be felt, and nodules frequently occurred in peri-hepatitis and in syphilitic liver. Moreover, a carcinomatous liver might be of huge size without having any nodules at all. He showed the picture of such a case lately under his care, in which there were no nodules, the condition being endothelioma. Therefore nodules were not to be relied on as of any diagnostic importance. Where there was a difficulty in recognition it would be well to give mercury and iodide of potassium first, and leave the question of exploration until afterwards if that were necessary. Three cases were shown at a meeting of the Clinical Society of gummatous enlargement of the liver, in which the abdomen was opened and the liver seen to be gummatous; the abdomen was closed and the patient put on iodide of potassium and mercury. In those cases he thought the iodide of potassium should have been given first. Generally the person with a syphilitic enlargement of the liver was in better general health than the person who had a cirrhotic liver. With regard to cases of cancer of the liver in which there was jaundice or ascites, one never had jaundice without bile in stools in carcinoma of the liver without ascites being also present. Jaundice was no part of carcinoma of the liver; when it occurred it was an accident, due to pressure of enlarged glands on the portal fissure. And if these glands were sufficiently enlarged to compress and obstruct the bile-duct, they were sufficiently large to press on the larger and softer portal vein and cause ascites. If there were *obstructive* jaundice and no ascites it was not carcinoma of the liver. He knew that was contrary to what was stated in the books. A large liver with obstructive jaundice but without ascites was usually due to carcinoma of the pancreas, and not to cancer of the liver. He thought in carcinoma of liver ascites very rarely occurred without jaundice, and ascites alone or obstructive jaundice alone with a big liver almost excluded cancer of the liver as a cause. Where ascites occurred with carcinoma of the liver and there was no jaundice it was nearly always cancer in the peritoneum or in some other internal organ. When both obstructive jaundice and ascites were present the diagnosis was difficult, but the presumption was that it was not carcinoma of the liver. He thought the latter was diagnosed more frequently than it occurred. He had seen two or three cases of influenza with hepatitis, one of which, in the first influenza epidemic, deceived him very much. A woman after an influenza attack had a huge painful liver, and he thought it must be carcinoma. But the enlargement entirely cleared up, and was due to acute hepatitis. Dr. Rolleston's observations about enlargement of cervical glands should be carefully borne in mind, because in doubtful diagnosis it was a positive symptom of importance.

Dr. CHAS. W. CHAPMAN asked whether the author had come across an enlarged liver in connection with malaria which was mistaken for malignant disease. Many years ago a gentlemen residing in Bristol was very ill, wasted, and jaundiced. The general opinion of physicians in Bristol was that he had malignant disease of the liver. He came to London and saw Sir Richard Quain, who on close questioning found that he had at one time had malaria. He was given quinine, and got perfectly well and put on flesh. He had since twice held the position of Mayor of Bristol.

Dr. POYNTON said the hydatid thrill mentioned in books in connection with hydatid disease of the liver was when present an important sign, and he recently came across an extraordinarily good example. This thrill was unlike anything else, and enabled him in his case to arrive at a diagnosis without wasting time, although there was a suspicion of syphilis. The diagnosis proved to be correct. Occasionally there were pedunculated

growths in the liver, and one such case he saw simulated gall-bladder, and the patient was operated upon under the idea that it was a gall-bladder disease. He thought such cases must be very uncommon. Another condition of things in which diagnosis might be very puzzling was in children in whom there was multiple serositis, with adherent pericardium and chronic inflammation of the pleura and great enlargement of the liver. The large liver might be thought to be the result of growth. In children also there occurred large tumours springing from the supra-renal gland on the right side which might push forward the liver, or might be associated with growths in the liver. Another question was the occurrence in adults of chronic tubercular disease. When the mesentery at the lower margin of the liver and bowel became much infiltrated with chronic tubercle, it gave one the impression that the liver was enlarged and nodular. In some of those cases it was not easy to tell the condition from malignant disease of the liver, and it was, still more difficult to distinguish them from syphilitic peri-hepatitis with gumma.

Dr. VINCENT BELL asked whether Dr. Rolleston had seen cases which appeared to be cirrhotic in the first instance, and which then showed a tendency to become carcinomatous.

Dr. L. GUTHRIE said he had been interested in the remarks on pyrexia in association with malignant disease of the liver. He did not think that was generally recognised as a symptom, and until fairly recently it was new to him, until he saw a case which puzzled not only himself but those who saw it with him. He was asked to see a lady, æt. 45 years, because she had pyrexia which had been going on for nearly six weeks. The temperature was spiky, and always began to rise at 4 o'clock in the afternoon, and usually reverted to the normal at 8 o'clock in the morning. It seldom rose beyond 102° F. She had been travelling in the Mediterranean in the early part of the year. In August she had what was supposed to be an ordinary attack of influenza. She recovered fairly well, but was not quite well, and she was sent to the country, where, however, she became worse, and her temperature was up. Accordingly she was sent home. When he saw her in November the temperature had been going up for five or six weeks in the evenings. During the preceding ten days she had had slight jaundice. Tongue furred, abdomen soft; there was no sign of ascites. The liver could be felt two and a half fingers below the costal margin. Blood-tests for Malta fever and suppuration were negative, but Widal's reaction was positive, so it was considered the case might be one of typhoid. A few weeks later the temperature fell gradually and for three or four weeks remained normal. She had constipation alternating with diarrhœa, and the motions were clay coloured and sometimes pea-soupy, resembling very strongly enteric motions. She did not improve, the jaundice did not increase, the liver enlarged slowly but was not painful, but there was constant nausea and vomiting. Eventually she died. Post mortem, encephaloid carcinoma was found infiltrating almost the whole of the liver, and the primary growth appeared to be in the common bile-duct. He thought the point about the pyrexia was a very important one, and those who were not acquainted with it might easily go wrong, as he did and those who were associated with him in that case. The use of anti-syphilitic remedies in the case of gumma of the liver had been mentioned, but in children he had not seen them produce any good effect. He had always regarded syphilitic liver in children as a particularly fatal disease.

Dr. ROLLESTON, in reply, said he had met with spasmodic dysphagia in cases of carcinoma of the stomach, and since gastric carcinoma was the commonest cause of secondary malignant disease of the liver it might easily happen that spasmodic dysphagia reflex from the stomach might be associated with a malignant liver. He did not know of the occurrence of reflex spasmodic dysphagia in primary malignant disease of the liver. It certainly happened that in some cases anti-syphilitic treatment failed to modify hepatic gummata, and that after laparotomy and removal of some of the gummatous material renewed anti-syphilitic treatment was successful. It was probable that in those cases either the gumma were very large, or that secondary bacterial infection of the gummata had taken place, and that removal of some of this gummatous material enabled the specific remedies to act at greater advantage. The occurence of pedunculated malignant growths of the liver was certainly very rare, but he had seen a pedunculated growth attached to the liver in generalised melanotic sarcoma. Secondary carcinoma in the liver might be confined to a constriction or Riedl's lobe of the liver, and in most exceptional instances primary carcinoma might start in such a tongue-shaped lobe. Primary carcinoma did, in a certain proportion of cases, arise in a cirrhotic liver. This condition, called "primary carcinoma with cirrhosis," was interesting as an example of malignant disease arising in a chronically inflamed organ, a sequence comparable to carcinoma supervening on chronic superficial glossitis or on chronic mastitis. Dr. S. Phillips' remarks as to the concurrence of jaundice and ascites in malignant disease of the liver, and his generalisation that a morbid enlargement of the liver accompanied by one only of these symptoms was almost certainly not malignant disease, seemed to be a very valuable and original contribution to the subject.

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WITH MR. BLAND-SUTTON AT MIDDLESEX HOSPITAL.

GENTLEMEN,—In the next four or five lectures which I have to trouble you with I propose to deal with the subject of cancer. To-day I shall devote the lecture to the general consideration of the subject, to cancer in a general way, and in the succeeding lectures I shall treat of cancer as it affects separate organs of the body.

First of all it is necessary for us to consider what we mean by cancer. Cancer belongs to an extraordinarily interesting group of diseases, which surgeons label malignant disease; and by malignant disease they mean a disease which, no matter what organ or part of the body it occurs in, will, if left to itself, inevitably destroy life. Now, these malignant diseases are of great interest, because they may indeed arise in any tissue of the body. You know from your anatomcal studies that the body is made up of two great groups of tissues, one known as the connective tissue, and the other as epithelium. In addition we must make a separate group of the two important tissues of the body, striped or voluntary muscle fibre, and nerve tissue. The connective tissues include all such things as fibrous tissue; it does not matter whether it is the stout stuff which forms the periosteum of bone, or the delicate tissue which forms the sustentacular framework of the retina, which is so fine that it requires special preparation to show it, even under the microscope. Then there is the coarse stuff known as bone, and gristle or cartilage, and fat. All these things are known as connective tissues, and they give rise to tumours, each of which receives an appropriate name, according to the kind of tissue in which it arises. Even amongst these connective tissue tumours there is a group which is malignant, and known as the sarcomata. The next group, the epithelial element, is of very

great interest. The epithelium of the body serves two purposes. In certain parts of the body it is protective, *e.g.* where it covers the surface of the skin, and the interior of the mouth and lips. In these situations it is of a characteristic shape, and it is usually spoken of as squamous epithelium. But where the epithelium permeates the organs and dips in to form secreting structures, such as the mammary glands, the sweat glands, or the liver or kidneys, and other complex organs, including the epithelium which covers the whole of the alimentary canal from the lips to the anus, and which dips beneath the surface to form the crypts of Lieberkühn and the other secretory glands in the intestine, it is more or less columnar in shape. Any of this epithelium may take on, from some cause, we know not what, malignant action, and form a tumour which will spread and destroy life. These malignant tumours are known as cancers. These cancers may be, or should be, described as malignant tumours arising in epithelium. And when they arise from the surface of the body, from the squamous-celled epithelial structures, we speak of them as squamous-celled cancers. When cancer arises from the epithelium which forms a secreting gland it is spoken of as gland cancer. You will find, when you come to read your text-books, that a squamous-celled cancer is often spoken of as epithelioma. That is not a very strict term, because all cancers are epitheliomata in the sense that they arise in the epithelium. And so, in order to avoid confusion, it is better not to speak of cancer arising in squamous-celled structures as epithelioma, but to always speak of it as squamous-celled cancer. And in that way, when you read monographs written by Germans or Frenchmen, it will save you much confusion at the outset. There is another part of the body where the structures are lined by flattened cells, which some years ago led to a good deal of dispute between physiologists as to whether they belonged to the connective tissue group, or whether they belonged to the epithelial; I refer to those peculiar cells which line the interior of blood vessels and lymphatics and the serous surfaces, such as the pleura, peritoneum, and the pericardium which constitute the endothelium. It is now known that endothelium in any situation may at times take on malignant characters and form tumours, which will probably destroy the individual who is unfortunate enough to grow one.

There is another peculiar kind of epithelial tissue in the body which only exists for a short time, and that is the peculiar epithelium which covers the surfaces of the chorionic villi, those delicate structures out of which the placenta is formed. These villi in the early stages are made up of an axis of delicate connective tissue, overlapped by a peculiar cell-mantle, possessing remarkable erosive properties, which enable it to penetrate living organs. It has been discovered lately, that is to say in the last few years, that this remarkable epithelium which covers the chorionic villi may, under certain conditions, become malignant, particularly when it undergoes the peculiar change known as hydatidiform degeneration. These cells may form a malignant tumour, and burrow through the uterus, make a large tumour inside the muscle walls of the uterus, may permeate the blood-vessels, and, becoming disseminated by the blood-vessels, destroy the patient. This is a very remarkable disease, and was originally termed deciduoma malignum, because it was supposed to arise in the decidua. Now it is spoken of as chorion epithelioma. It is not a very good term, but it identifies the disease with the tissue in which it arises. When we come to deal with it specifically I think you will agree with me that it is one of the most extraordinary forms of malignant disease which attacks the human body.

But that does not dispose of malignant disease. You know that the ovary contains in each ovarian follicle a cell which is known as the sex-cell, and when the environment is favourable it may grow up and become an individual, that is to say, when the egg is fertilised. Under certain conditions these sex-cells in the ovum may take on a malignant action and produce a tumour in the ovary, even in children in the early years of life. This tumour behaves exactly like a malignant tumour, and destroys the children in whom it occurs even before they pass out of the period of infancy. These are known as malignant teratomata. So that even from the ovum to the mature tissues, for reasons of which we know nothing, malignant tumours may arise and destroy the individual. There are five malignant groups formed in this way. First of all the typical carcinoma or cancer. Then we have the variety which is derived from the endothelium, called endothelioma. Then we have he chorion-epithelioma coming into the third

t

group. These are bracketed together because they are all derived from the epithelium. Then we have those which start in the connective tissue, the group of sarcomata; and, last of all, there are those which start in the ovum or egg, the malignant teratomata.

We shall, in the course of the next few months, have to deal with examples of each of these groups; but to-day I shall draw your attention more especially to the commonest of all these—namely carcinomata; these arise in the epithelium of the adult body. We divide carcinoma into two great groups; those which arise from squamous cells, and those which arise from the epithelial cells of glands. A carcinoma may arise in any part of the body where there is epithelium, and there are certain conditions which predispose to the starting of a cancer. For instance, on the exterior of the body it is extremely common at the margins of the lips, and on the tongue, and at the margins of scars left by burns, and in old ulcers. It may, perhaps, even attack an old wart. A person may have had a solitary wart on his body for many years. He does not worry about it, and then towards middle life, perhaps, this wart becomes painful, and may even ulcerate. Perhaps his clothes rub it, or his braces, or his garter, or some article of his clothes, and then it will become cancerous. The same may take place in the lip. He may be a man who is accustomed to smoking, and at the place where he puts his pipe between his lips a little crack forms in the mucous membrane, and this will ulcerate. In due course the lymph glands in the neck enlarge, then by degrees the skin ulcerates and forms deep holes in the neck. In time the ulceration will open up the blood-vessels in the immediate neighbourhood, and the whole thing will form a huge ulcer extending from the lip to the neck. Of this you will see plenty of examples in the hospital. They are always here, in all stages, and in their final stages you will see them in the Cancer Wing. The most insidious way in which these cancers arise is when they form in the interior of a gland, like the breast. Now, in the breast the patient probably will not feel any pain, but she will notice on looking at herself that one breast looks a little different from the other—perhaps the nipple is a little drawn in, or the skin may be dimpled over it, and there is a little alteration in the contour of the breast. Then she feels it, and finds there is a small hard lump in the breast. This lump which forms in the breast is spoken of as the primary focus of the disease, or the primary cancer, and, in the early stages, if you take one of these lumps after it has been removed from the body, and make sections through it, you will find, as a rule, that in the early stages it is quite hard, so hard, indeed, that when this disease was first known these hard lumps in the breast were spoken of as scirrhus. It used to be called scirrhous cancer, because it was so extremely hard. And then when you make a section through one of these lumps in the breast you see a portion of it looking more like the cut surface of an unripe pear than anything else. And when you look at it carefully you cannot tell exactly where this hard stuff ends in the breast, for it passes indefinitely into the soft tissues of the breast. This indefiniteness is a most important thing with regard to cancer, because in nearly all the innocent tumours which you will have to deal with, you will find when you divide them, that the tumour itself is enclosed in a soft sac which is known as a capsule, and this capsule isolates it from the surrounding tissues. And you can, in the case of these encapsulated tumours, make an incision into the capsule and turn out the tumour as easily as you can turn a ripe banana out of its rind. But this is not the case with cancers. A cancer is an indefinite swelling in the breast; you cannot, with your naked eye or with your fingers or with any of the methods which we use in the ordinary way of surgery, tell the limits of that growth, and you cannot always be sure of defining the limits of the growth even when you have examined it under the microscope. This peculiar lack of limitation of the growth is one of the most characteristic things in regard to cancer, and it is, of course, one of the most dangerous things in connection with it. When you prepare a section from the nodule in the breast, and stain it properly and examine it under the microscope, you will find in all of them much the same characters. You will be able, under the microscope, to recognise two tissues, the parenchyma and the stroma. The stroma is made up of fibrous tissue, and the section is divided by it into alveoli, and each alveolus or hollow is filled up with epithelium. And the peculiar point about the epithelium of the cancerous growths is this; that in whatever gland the cancer arises the epithe-

lium which is found in these alveoli is characteristic of the gland in which the cancer arises. If it arises in the breast, these cells will have the characteristics of the ordinary cells of the mammary gland. When it arises in the uterus, these cells will be columnar, exactly like the columnar epithelium which lines the interior of the uterus. If it grows in the liver it will have the same kind of structure as the liver cells, and if it grows in the bile-ducts it will have epithelium of the same character as that of the bile-ducts. If it grows in the thyroid gland it will have the same structure as the thyroid body. Indeed, so characteristic are the cells of cancer that if a carefully prepared section is put before a competent morbid histologist he can, with almost absolute certainty, tell you the part of the body in which the particular cancer arose. I remember many years ago seeing a very striking example of that in this hospital. There was a man in the ward who had some trouble with his abdomen, nobody could quite be certain what. But in a little while the whole of his skin became dotted over with small knots, and then we knew that he was suffering from dissemination of cancer. And as it was important to know the primary source, one of these little nodules was excised from the skin, and when examined under the microscope it showed all the characters of the peculiar gland found at the pylorus. We therefore knew at once that in all probability he had a cancer of his pylorus. As time went on, and he eventually died, cancer was found in his pylorus. It is only one example of many; you will have plenty of opportunities of seeing all these things demonstrated in practice. I want you to first understand with regard to the primary tumour, that this is one of the peculiarities of cancer; and this is what is the puzzle to pathologists at the present day, that whatever gland the cancer starts in the epithelial cells of the growth correspond exactly to the cells of the organ in which the cancer takes its rise.

We have now to consider the stroma. The nature of this tissue has been particularly investigated recently, and especially in the cancer department here. The stroma which forms around these cells is probably a protective effort of the connective tissue excited by the activity of malignant epithelium. It is now the fashion to speak of this as the connective-tissue reaction. You know you may get a shot in your body, or a piece of needle in your finger, or some such thing. If the foreign body is clean, the tissues will tolerate it; but they throw round that foreign body a capsule of connective tissue. You can study that in the peritoneal cavity. It sometimes happens that we often swallow with our food foreign bodies, such as a minute piece of millstone perhaps, a small fragment of a pot, or even the point of a needle or of a pin. If that makes its way through the bowel into the general peritoneal cavity and it is not septic, this little piece of metal will cause a good deal of irritation, and then some fluid and cells known as lymph will be thrown out around the body, and it will become embedded in stuff like so much gum. In the course of time this exuded material will be organised into fibrous tissue, and by degrees if it remains there for a long time it will be encysted and form the nucleus of a round ball of organised fibrous tissue, and thus be rendered innocuous. The same thing takes place around the advancing columns of cancer. As they grow down into the connective tissue they produce what is called a connective-tissue reaction, and these columns become surrounded by a sort of capsule of connective tissue, and as each column gives off other buds these columns become thrown out in different shapes and forms from the parent column, and they will become embedded and buried in this connective-tissue stroma. As a rule primary cancers do not grow to any great size; they remain quite small, rarely in the breast do they reach a size larger than an orange, often not larger than a chestnut. They rarely attain any large size because they are but poorly supplied with blood-vessels, and the tendency of them is to shrink, and the connective tissue which forms around the epithelium columns becomes very firm, and causes the epithelial cells to shrink. Sometimes the tissue contracts so firmly on the columns that the epithelial cells undergo changes and become absolutely destroyed, and the cancer will undergo spontaneous cure. There is no doubt that occasionally in the breast and other parts of the body, certainly in the small intestine, some of these primary foci of cancer will get stifled by the connective tissue, and spontaneous cure is the result It is this growth of connective tissue which makes the tumour so hard. But although this primary focus undergoes spontaneous cure, it nevertheless

will destroy the patient's life in another way. All the malignant tumours are peculiar in that they disseminate cancer through the body. After a primary focus has existed for a few months the cells will implicate the lymphatics which are contained in the connective tissue, and make their way to the nearest lymph glands. When you are learning anatomy in the dissecting-room you rarely perceive how important the lymphatics and lymph glands are; as a rule the students neglect them in the routine dissecting-room work.

When you work at surgery and attempt to make a diagnosis in the wards you will find these lymph glands are of the utmost importance. In the case of cancer in the breast, the cancer cells find their way into the mediastinal lymph glands, the axillary gland, and then creep up and affect the glands in the neck. They find their way into the thoracic duct, and flow into the veins, and from the veins they are conveyed into the heart, and from the heart they are pumped through into the lungs. The lungs, as you know, are permeated by small blood vessels, the pulmonary capillaries; and in these pulmonary capillaries these small cancer cells are strained from the blood, and there they implant themselves on to the tissues of the lungs and form what are known as secondary cancers, nodules, or knots. And the number of these secondary knots which may be found about the body is extraordinary, two or three hundred or more of them. Under certain conditions if these cancer cells find their way into the left side of the heart, as they do occasionally, in the way in which I shall explain to you later, these cancer cells may be distributed all over the body by the blood and produce secondary knots in every organ and every tissue of the body, not only in the lungs and liver and soft parts of the body, but they also find their way into the bones.

We believed until recently that these secondary knots were entirely due to the dispersal through the body of cancer cells by the blood stream, and the appearance of secondary knots in the body of cancer is known as metastasis. Until lately it was thought that these cancer cells were distributed through the body by the blood stream, like an embolus. This is known as the embolic theory. But since then Mr. Handley has been working very hard at this subject, and he shows that these secondary knots in the bone, although some may be due to embolism, the majority are due to quite a different process. He has discovered that when, for instance, in the breast, for that is the simplest case, a cancer forms, the cancer cells creep into the lymphatics of the breast and spread all round it, and then find their way into lymphatic plexus in the fascia which covers the thorax, and then the cancer cells spread through these lymphatics and creep into the lymphatics of the arm, into the pleura and affect the lungs, and that then they make their way towards the epigastrium, and find their way along the fascia of the abdomen, reaching the fascia of the limbs and periosteum of the bones. The cancer spreads from a central spot in widening circles until at last it gets all over the body, and, if the patient lives long enough, it may even reach the hands and feet. This remarkable process, the truth of which Mr. Handley has practically established, is called permeation. It is quite certain that, although this theory of permeation explains the greater number of cases of metastases, nevertheless it is true that cancer is also disseminated by means of the blood-vessels, but in a minor degree. Some interesting work has been done in that way by Schmidt, of Vienna. He made some very interesting observations on the fate of cancer cells in the blood. If cancer cells really are distributed through the blood-vessels, surely if we examine the blood of a cancerous patient where dissemination is going on we ought to find cancer cells in the blood. I have spent a good deal of time and much hard work in trying to find out whether we could find these cells actually in the blood, but I have never seen one in my life, but Schmidt, of Vienna, went to work and examined the veins in the lungs in cancer patients. Here is a picture of a section of the pulmonary vein, and it shows the particles of cancer in it. So here is a positive proof that the cancer cells do get into the blood stream. But when the farmer sows wheat over his field every grain that he sows does not come to maturity. So it is here. Schmidt showed that the blood has a defending power against these cancer cells. Here is a vein in transverse section with a cancer embolus in it. Schmidt shows that when a piece of cancer got into the blood stream, the blood clots around it. When a thrombus forms in the blood stream, it excites thrombosis and the clot isolates it from the blood stream. These cancer cells undergo degene-

rative changes, and they became inert and unable to engraft themselves on to the tissue. This is interesting too in another way, because as you know quite well, whenever you find a secondary knot in a patient you know quite well, although you may not know where that primary tumour is, that it will be of the same structure as the primary tumour. And that has always been a puzzle about these secondary knots, they are found to be always of the same nature as the primary focus of disease. I remember not very long ago a very interesting case, where a woman had a lump in her back, and it was thought to be a primary tumour. The tumour was excised, and on being examined microscopically it was found to have all the structure of the thyroid gland. Her thyroid gland was examined and it was found that she had a big thyroid, and that the tumour from the back was secondary to one in the thyroid body. And I was very much impressed many years ago by seeing a case shown at one of the societies where a woman had a tumour as big as her head, growing from the shaft of the humerus. It was thought to be sarcoma growing from the periosteum at the upper extremity of the humerus. But after it was removed the pathologist microscoped it and found it was not sarcoma, that it was lined with tall columnar epithelium, and had lymphoid follicles similar to those in the rectum. Then on examination it was found that she had a primary carcinoma in her rectum. You will find many extraordinary things like this. It shows that these secondary knots are due to the actual transference of small particles of primary growth which find their way into the blood vessels or into the lymphatics. They are then broken off and discharged into the blood stream and carried as emboli and grow into tumours, or they may of course be conveyed by the process of permeation along the lymphatics.

To show you how these cancer cells may be discharged into the circulation by the thoracic duct I show you this remarkable specimen; it is a thoracic duct from a patient who had cancer of the stomach, and these are the lymphatic glands at the posterior part of the abdomen and parts about the receptaculum chyli. Many a student has dissected a thorax and taken the existence of the thoracic duct simply on the word of his teacher; he had never found it himself because it was so thin and delicate. In this case the primary cancer of the stomach infected the lymphatics about the receptaculum chyli, and eventually converted the thoracic duct into a solid cord, and infected the lymph glands lying in the left posterior triangle of the neck. You can imagine the large number of cancer cells which must have found their way into the blood in such a case. And it is interesting in another aspect, because some of you know well that when you are examining a patient with suspected carcinoma of his abdomen, or a suspected tumour of the stomach, and you are not sure what it is, you will see the physician or the surgeon, as the case may be, feel at the root of the patient's neck in order to ascertain if this patient has an enlarged gland in that situation.

Many years ago we were puzzled to know how it could be when a man had carcinoma of the stomach he should get an enlarged gland in the root of his neck; although we could not explain why it enlarged, we know it to be a very good diagnostic sign. But here is an explanation of it. The glands in the abdomen become enlarged, the thoracic duct becomes infected with carcinoma, and conveys the cancerous particles to the gland in the root of the neck. There is another point which you must disabuse your minds of. The idea you have is that cancer is, as a rule, quickly fatal. As a matter of fact, cancer itself is a very slow and very chronic disease. Cancer may take years to kill a patient, just as leprosy may. I spent last Christmas-day in the Leper Hospital at Jerusalem, and there was a man in that institution who had had leprosy for thirty years; and I have seen patients in this hospital who have had cancer twenty years, and you will hear of many cases of cancer which have persisted for a long time. It is not the cancer which kills; it is the accidents to which that patient is liable; it is these secondary growths, they block up the lung and get into the brain, and they block up the blood-vessels. Or, if they are in situations where the air has access to them, micro-organisms get in, and they begin to ulcerate in consequence, and they decompose and slough.

Here is a uterus which, during life, was three times its ordinary size. It has walls almost as thin as a bladder, and it is filled with a large lump, which you might think is a fibroid. It is really a mass of cancer. The patient was a spinster lady who had never had any children. The point of

that remark is that, if the patient had been fertile, if she had been a married woman and borne children, the cervix would have been open and would have furnished a highway for micro-organisms. But in women who have never had children the opening of the cervix is very minute, and no micro-organisms gain entrance. Therefore, when there is a mass of cancer like this, that cancer grows undisturbed by micro-organisms, it attains huge proportions. I have seen them in the ovary in the same way, weighing several pounds. So if you have a patient who has carcinoma and the air cannot get at it, and there is no ulceration and no infection, that patient may go on and live for very many years. But directly micro-organisms gain entrance to it they become septic, there is decomposition, sloughing, and infection. Usually the patient dies from some terminal infection like pyelitis, septic pneumonia, peritonitis, or some condition of that sort. It is not the cancer itself as a disease which kills, it is the secondary conditions associated with it, such as sepsis, pressure on important organs, and especially the mischief caused by the dissemination of cancerous particles through the body, and the production of secondary deposits.

December 14th, 1908.

Treatment of Carbuncle.—Grasmann says the quickest and surest treatment of carbuncle consists in extirpation of the diseased area by a circular incision with smaller incisions radiating from it. Such incisions should in no case be carried through the actively inflamed portion of the lesion, but should be placed in the infiltrated skin surrounding it. The whole carbuncle should then be separated from the underlying fascia and the large wound packed with dry gauze. In other cases cross incisions through the carbuncle may be used, the four flaps thus made being lifted from the underlying tissue and supported by gauze packing; necrotic masses are to be removed with the knife or the scissors. After a few days, when the granulation tissue has well developed, the flaps are brought together by a couple of stitches, thus diminishing the size of the wound. The latter is completely closed within four to six weeks.—*Medical Record*, vol. lxxiv, No. 21.

ACUTE LYMPHOCYTHÆMIA IN CHILDREN.

A Clinical Lecture delivered at the North-East London Post-Graduate College.

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I PROPOSE to-day to speak about a rare form of blood disease which is not as yet sufficiently recognised as to find its way into the ordinary text-books. I refer to acute lymphocythæmia with no, or at most only a very moderate, leucocytosis. By lymphocythæmia is meant a great preponderance of those mononuclear cells in the blood which go by the name of lymphocytes, amounting in some cases to the almost total exclusion of all the other forms of leucocytes. The disease is apparently a variety of what is usually known as lymphatic leukæmia, though the term "leukæmia," or "leucocythæmia," usually implies the existence of a very marked and evident leucocytosis. Of leukæmia, you will remember, there are two forms—the myelogenous and the lymphatic. The former is characterised by a chronic course, a great enlargement of the spleen and an anæmia consisting of a moderate reduction in the number of the red corpuscles, among which erythroblasts, or nucleated red cells, are very commonly seen; a great increase in the number of leucocytes up to 400,000 or 500,000 or more per c.mm., of which myelocytes form about 30 per cent., while the eosinophile cells are present in an increased proportion. Of this form of leukæmia I shall not speak further to-day, as it is beyond the scope of my lecture. The lymphatic form of the disease is generally divided into chronic and acute varieties. The enlargement of the spleen is not so marked as in myelogenous leukæmia, but the lymphatic glands throughout the body usually show a marked increase in size. The chief points about the blood are a considerable diminution in the number of the red cells, the scarcity of the nucleated forms except when the disease is acute, and a leucocytosis of about 100,000. Of the white cells the lymphocytes constitute over 90 per cent., and, speaking generally, the small lymphocyte

predominates in the chronic form of the disease, while in the acute variety it is the large cell that is in excess. In lymphatic leukæmia the eosinophiles are very scanty.

I have thus given a very brief survey of what is generally understood by leukæmia, but I wish to impress upon you that the diseases of the blood show a greater variety than the typical forms of leukæmia which I have just described and such conditions as chlorosis and pernicious anæmia, with which we have nothing to do to-day. The more the pathological condition of the blood is investigated the more complicated the subject seems to become, and you may take it that, speaking broadly, no two cases of blood disease show precisely similar features.

I now come more especially to the subject about which I wish to speak, and I will premise by saying that I prefer to give the disease, or at least this particular variety, the name of "acute lymphocythæmia," a name which is at once comprehensive and sufficiently indicative of the condition. It is a disease which is confined, as far as my experience goes, to early life, and runs a rapid and fatal course.

The chief characteristics are progressive anæmia and weakness, and enlargement of the spleen and liver and of the lymphatic structures generally. There is a tendency to hæmorrhages, and the temperature is raised. The white blood-corpuscles consist almost entirely of lymphocytes, and these are for the most part of the large variety. The total number of leucocytes per c.mm., however, is only moderately, if at all, increased.

With these few introductory remarks I will read to you the notes of three cases which have come under my own observation, and will afterwards deal with the more important points concerning the condition in greater detail.

Case 1.—A girl, æt. 2 years, came under my care in August, 1905, with a history of four days' fretfulness, malaise, and constipation. The inguinal lymphatic glands had rapidly become enlarged and painful, and on this account the child had been scarcely able to walk. She had had bronchitis and measles a year previously. She was thin, fretful, and of a sallow complexion, but not anæmic. The cervical, axillary, and inguinal glands were all moderately enlarged, but no increase in size of the liver, spleen, or thymus could be made out. The temperature was a little raised, and by the end of a fortnight the glands had increased in size and fresh ones could be felt. The spleen also was just palpable below the ribs, and the liver edge extended to nearly halfway between the costal margin and the umbilicus. The glands continued to enlarge, and subsequently the patient had some bronchitis and a severe attack of herpes zoster, which was accompanied by a temperature of nearly 105° F. The anæmia gradually became more marked, and hæmorrhages occurred, both in the skin, in the shape of purpuric spots, and from the lips and gums. The child rapidly sank and died three months after the onset of the symptoms, the temperature at the end varying between 102° and 104° F. The blood was examined on several occasions, and showed a rapid diminution in the number of red cells. The whites varied considerably from time to time, the highest count being 25,000 and the lowest 5600 on the day before death. The lymphocytes were about 90 per cent., of which by far the greater number were of large size. Nucleated red cells were scanty, and the colour index was slightly lowered, showing a certain deficiency in the amount of hæmoglobin present.

Case 2.—While the first case was under observation, another girl, æt. 4 years and 8 months, was brought to me on account of general pallor, which had been noticed for about three weeks. Two weeks previously a purpuric rash had appeared, and the child has been occasionally sick, the vomit being streaked with blood. There was a family history of rheumatism, and the patient herself had had measles when two and a half years of age, and diphtheria six months previously to her present illness. The child was very blanched, and a diffuse purpuric rash covered the trunk and extremities. One hæmorrhage was also present on the lower gum, and another in the left fundus oculi. The cervical, axillary and inguinal glands were all enlarged, but not tender. In the lungs there were signs of bronchitis, and the heart was considerably dilated. The spleen was much enlarged, extending down to within three inches of the centre of Poupart's ligament, and was soft but not tender. The liver was also large, soft and painless; the lower edge was four inches below the ensiform cartilage. Blood-streaked vomiting occurred from time to time. The bronchitis became worse, and the temperature varied between 100° and 102° F.,

the child finally dying within four weeks of the onset of the symptoms. The red blood-corpuscles were less than half the normal quantity, while the leucocytes varied between 17,000 and 20,000. The lymphocytes on one occasion amounted to 98·4 per cent. of the total leucocytes, about 72 per cent. being of the large variety. Nucleated red cells were very scanty, and the hæmoglobin value was good, being only just below normal.

CASE 3.—In May last a boy, æt. 7 years, was admitted to the Evelina Hospital under the care of my colleague, Mr. Clogg, for enlarged glands in the neck, axillæ and groins, which had been first noticed six weeks before, and had rapidly increased in size, while the child's general health had deteriorated. There had been some temporary swelling and pain in various joints, but these had disappeared with rest. The weakness, however, persisted, and the temperature had fluctuated considerably. On one occasion there was blood-stained vomiting. The boy was very pale and anæmic, and had large masses of glands in the neck and in both axillæ and groins. The glands were firm and discrete, and showed no signs of softening or of suppuration. There were also signs of enlarged glands in the mediastinum. The splenic area was tender, but the spleen itself could not be felt, though its dulness seemed to be increased. A little later the spleen was palpable, and the liver became slightly enlarged. The glands further increased in size, and blood-stained vomiting occurred on another occasion. A trace of albumen was found at times in the urine. The patient rapidly went downhill, falling into a condition of drowsiness which alternated with delirium, and finally died in less than nine weeks from the commencement of his illness. The blood showed a marked diminution in the number of red cells, there being 1,510,000 per c.mm. on admission, and only 1,040,000 on the day before death. The leucocytes numbered about 6000, and of these the mononuclear elements amounted to roughly 90 per cent., of which the large size predominated as usual. A very few normoblasts and one megaloblast were seen, and the hæmoglobin value was about normal.

As regards the post-mortem findings in these cases, they all presented very similar features. The lymphatic glands in the deeper parts, as well as the superficial ones, were found to be enlarged, and many of them were hæmorrhagic. The thymus in two at all events was enlarged. Hæmorrhages were found in various organs, such as the heart, lungs, spleen and intestines. Microscopically the lymphoid elements were increased generally in the glands, spleen, intestines and marrow, and infiltrations of lymphoid cells were found in the various organs, *e. g.* in the heart muscle, in the portal spaces of the liver, in the spleen-pulp and in the kidneys. The pathological investigations thus fully confirmed the clinical diagnosis.

I now propose to go a little more fully into the details of the disease. It is, as I have already told you, a rare one. True lymphatic leukæmia is by no means a common condition, but these cases with only a slight leucocytosis, or even none at all, are still less frequently met with. My first two cases occurred in 1905, and it was not until the middle of this year that I came across another, though I have been constantly on the look-out for them. They are limited as far as my experience goes to childhood, and occur for the most part apparently during the first four or five years of life. The *sexes* are attacked about equally. The *onset* of the disease is fairly definite. Enlargement and slight tenderness of the glands are accompanied by anæmia and general weakness, which rapidly become more marked. In very rare cases the glands show no evidence of being affected, but as a rule there is a general enlargement. The superficial groups, such as the cervical, axillary and inguinal, stand out as definite masses. The individual glands are firm and fairly discrete, and show no signs of caseation and suppuration. At times they are tender, and so hinder the natural movements of the body and limbs. After death other groups are found to have been also affected; in the chest the bronchial and mediastinal glands are enlarged, as also are the mesenteric and retro-peritoneal in the abdomen. Hæmorrhages are a prominent feature; these occur in the form of a purpuric rash over the trunk and limbs, and as bleedings from the nose, gums, stomach or intestinal tract. Hæmaturia is rare, but retinal hæmorrhages are not uncommon. Vomiting is a frequent symptom, and diarrhœa may also be present.

The liver and spleen are usually enlarged, but to a varying degree—in some cases considerably, so that the edges of both can be felt some distance down in the abdomen, but in others only to a very

slight extent. A trace of albumen is sometimes found in the urine. The temperature is irregular and presents marked fluctuations, varying between 102° and 104° F. towards the end, with a corresponding increase in the pulse- and respiration-rates. The disease is a short and rapidly progressive one, a fatal termination occurring within three or four months, and in some cases even within as short a time as two or three weeks. Death may be brought about by a super-added bronchitis or pneumonia, or may be the result of increasing anæmia, weakness, and coma.

Diagnosis.—The true nature of the disease can only be determined by an accurate examination of the blood. In all suspected cases an estimation of the red and white corpuscles should be made, and also a differential count of at least 200 of the latter. As I have already pointed out, there is a considerable diminution of the erythrocytes, even down to about 1,000,000 per c.mm. Poikilocytosis and polychromatophilia are generally present, though not to a marked degree, while erythroblasts, or nucleated red cells, are to be met with in small numbers. Very rarely a megaloblast may be seen. The amount of hæmoglobin varies more or less with the number of red corpuscles, so that the colour index is usually not far removed from the normal. The leucocytes may show no increase in number, as in one of my cases, but, on the other hand, there may be a slight leucocytosis up to 20,000 or 25,000. The chief point about them, however, is that the mononuclear lymphocytes form 90–99 per cent. of the total number, the polymorphonuclear cells being very scanty indeed. The lymphocytes, for the most part, are of large size—a characteristic which is usually, though not always, true of acute lymphatic disease in general. In estimating the value of the fact that the lymphocytes are greatly increased in number, you must, of course, not lose sight of the fact that in early life these cells constitute a much larger proportion of the total number of leucocytes than they do in later years. Thus at birth the mononuclear cells form 50 to 75 per cent., and even as late as the third year lymphocytes and polymorphonuclears are present in about equal numbers. It is not until the eighth or tenth years that the proportions are those of the adult type.

The conditions most likely to be confused with acute lymphocythæmia are: (1) tubercular lymphadenitis; (2) lymphadenoma, or Hodgkin's disease (3) sarcoma in lymphatic glands. In tuberculous glandular affections the glands tend to be matted together and to break down and suppurate—features which do not occur in the condition under consideration. There are, moreover, no marked blood changes unless an abscess has been opened and infected, when an ordinary leucocytosis occurs. Lymphadenoma, on the other hand, very closely resembles lymphocythæmia in its external features. The glands present the same characteristics, and there is a concomitant anæmia, so that from a visual and tactile examination it is impossible to distinguish between the two. The course of the disease, however, is, as a rule, longer than that of acute lymphocythæmia. An examination of the blood will alone distinguish between them. In lymphadenoma a lymphocytosis does not occur until the later stages, and even then it is not nearly so marked as in lymphocythæmia. In sarcoma of the lymphatic glands also the blood-picture is different. There may be a slight leucocytosis, but the relations between the polymorphonuclears and the lymphocytes are unaltered.

Ætiology.—Various theories have been suggested as to the origin of lymphocythæmia. The one that has gained most favour is that the disease is due to changes in the bone-marrow, such changes being of the nature of a malignant growth. According to this view the invasion of the glands and viscera are really metastases. It must be remembered, however, that the number of lymphocytes in the blood varies considerably from time to time and does not show a steady increase up to the time of death, as might be expected if the process were of a true sarcomatous nature. Another theory is that the lymphatic glands are the seat of origin. This would account for the production of increased numbers of the small lymphocytes. Others have thought that the cause is to be found in a microbic infection of the blood. Streptococci have been obtained after death from the blood, spleen, and marrow (in my last case they were present in the marrow but not in the blood), but the possibility of contamination must be borne in mind. Occasionally, it is true, they have been obtained during life. The course of these cases, their rapidity and high temperatures render this view a plausible one. Possibly all the three theories which I have mentioned are concerned in the production of the

disease. A microbic or toxic infection may enter the blood and be carried by it to the lymphatic glands and marrow, in both of which certain processes, at present undefined, may be set up which give rise to an excessive formation of lymphocytes. These cells may then be driven into the circulation and deposited in the various organs, with the result that we get a condition of lymphæmia and lymphocytic invasion of the tissues such as I have described.

Post-mortem appearances.—Hæmorrhages are found in the skin and under the serous membranes, on the lungs beneath the pleuræ, on the heart under the epicardium, and occasionally on the surface of the brain; they may also be found in the retina. More extensive extravasations of blood occur in the lymphatic glands, in the tonsils, in the various organs, and also in the walls of the intestines. The tonsils are at times enlarged and may show deep crypts. The thymus tends to be persistent and large, and in Case 3 weighed as much as 14 grm. Microscopically there is often a proliferation of lymphocytes, both in the gland substance and in the connective tissue. The lymphatic glands throughout the body are swollen and enlarged, those chiefly affected being the cervical, axillary, and mediastinal, the abdominal and the retro-peritoneal, and the inguinal. Sometimes they are of a greyish colour, but at others red and soft. Speaking generally they are of a firm consistence and show no tendency to matting or to suppuration. Hyperplasia of the lymphoid elements is well marked, causing obliteration of the lymphatic follicles in most cases. The lymphocytes vary in size, and the large cells which occur in lymph-adenoma are not to be seen. The lymph sinuses are dilated, and these may be found packed with lymphocytes or in other cases empty. The connective tissue may show a certain amount of increase, and the endothelium may be proliferated. The spleen is generally more or less enlarged—in some cases decidedly so. The Malpighian bodies are ill-defined, and their centres may be occupied by granular structureless masses. The pulp is congested and crowded with lymphocytes. The connective tissue is slightly increased, and the capsule may show a certain amount of thickening. The liver is likewise enlarged and of a pale colour. Deposits of lymphoid cells are found scattered throughout its substance, in some instances limited to the portal spaces, but in others scattered diffusely both in and around the lobules. In places there may be small areas of degeneration of the liver cells. The kidneys are generally enlarged, and present marked lymphocytic infiltration, chiefly around the larger vessels and Bowman's capsules. In some cases the lymphoid deposits are so extensive as to leave very little normal tissue visible. The heart is often dilated, and shows fatty degeneration. Masses of lymphocytes are found between the fibres. The lungs are œdematous, and may contain hæmorrhages and patches of broncho-pneumonia. The stomach, as a rule, shows nothing abnormal beyond small hæmorrhages under the mucous membrane. The intestines at times contain unaltered blood with small hæmorrhages in their walls. The lymphoid patches show hyperplasia, the follicles being crowded with lymphocytes. The bone-marrow is pale and thicker than normal, but its colour may vary to a bright red. The lymphocytes are enormously increased in numbers, the majority being considerably larger than a red blood-corpuscle, but a certain number are of a smaller size. Granular cells are extremely scarce. Myelocytes and eosinophile cells are present, but in very small numbers. Comparatively few red blood cells are to be seen, and nucleated red cells are rare.

As regards the treatment of the condition, nothing, so far as I know, is of any real value. The disease is a virulent and a short one. I have treated my cases with arsenic and iron, but have not found that these drugs have been of any marked benefit. The general health should be attended to as far as possible, and stimulation will usually be found to be necessary.

December 14th, 1908.

Hot Air Treatment of Acute Inflammations.—Jselin applies the hot air the day after the abscess has been incised and evacuated and the cavity packed with iodoform gauze. He has thus been treating all phlegmons on the arms and hands during the last six months, and has been surprised at the way in which recovery was hastened, while the patients all lauded the hot air as having banished the pain. He uses the ordinary apparatus for using superheated air, applying it twice a day for two or three hours each time, maintaining a temperature of from 194° to 230° F.—*Journ. A.M.A.*, vol. li, No. 21.

WITH DR. ARTHUR WHITING IN THE WARDS OF THE PRINCE OF WALES'S GENERAL HOSPITAL.

THERE are two cases of muscular atrophy now in the hospital that I should like you to consider with me at some length this afternoon; the other cases in the wards we may look at more cursorily, but there is a third case of muscular atrophy—an out-patient—we will examine if there is time. Let us recollect what the types of muscular atrophy are.

There is a muscular atrophy seen in wasting diseases, such as phthisis and diabetes, which is a part of the wasting of the tissues of the body generally, and there are muscular atrophies which, at their commencement at any rate, involve a group or groups of muscles. It is hardly necessary to say that it is this second class of muscular atrophies—localised amyotrophy—that is, from our present point of view, the important one.

Muscular atrophy proper is either nervous in origin, when it is due to potential or actual disease of the grey matter of the brain-stem or spinal cord, and hence often called myelopathy; or essentially muscular, and called, therefore, myopathy.

First, a few words about the myelopathies. Under this heading may be gathered, for our purpose, not only those atrophies which result from a destruction of nerve cells in the anterior horns of the spinal cord and the motor nuclei of cranial nerves, but also those following disease or injury of the anterior nerve-roots, the motor nerve-trunks, cranial or spinal, and the terminations of the motor nerves in the muscles themselves. These we may class together, because in the last analysis the atrophy is the result of the cutting off, as it is believed, of the trophic influence of the motor nerve cells from the muscle-fibres, giving the same effect as if the cells themselves were destroyed. The main types are anterior poliomyelitis, a lesion of grey matter *en bloc*, giving, if acute, the rapid paralysis of children, the so-called infantile paralysis, or rather, less rapid in adults, or if not acute the subacute or chronic anterior poliomyelitis of adults; and progressive muscular atrophy, due to a lesion of the spinal grey matter cell by cell, commencing in the small muscles of the hands, and affecting adults. If the nuclei of the brain-stem are affected the condition is known, as you are aware, as bulbar palsy or labio-glosso-laryngeal paralysis. As a type of atrophy due to lesions of the motor nerves that occurring in ulnar paralysis may be taken, in which the wasting is chiefly shown in the hypothenar group, the interossei, and the ulnar portion of the thenar group. The ulnar nerve being a mixed nerve there is also anæsthesia. As a type of atrophy due to lesions of the peripheral terminations of nerves—a terminal neuritis—may be taken the atrophy of the anterior tibial and calf muscles in alcoholic neuritis. Here, again, inasmuch as the sensory terminations are affected equally with the motor nerve-endings, there is anæsthesia.

In primary muscular atrophy, on the other hand, there is no evidence whatever of any involvement of brain-stem, cord, or nerve; it is believed to be essentially a disorder of the muscular substance, and is hence called muscular dystrophy or myopathy.

One of the two cases for this afternoon is a myelopathy, and the other is a myopathy, and each is a somewhat rare example of its respective class. The myelopathic case is an instance of acute anterior poliomyelitis in adults, and the myopathic case one of the facio-scapulo-humeral type, often known as the Landouzy-Déjerine type, after those two distinguished French physicians.

Let us consider the case of acute anterior poliomyelitis first. Here is the patient, a single woman, æt. 30 years, a cook by occupation. Before, however, proceeding to read the history of her case, I should like to define the term "acute." Acuteness in these and other diseases is merely a matter of degree. The subacute cases of poliomyelitis may be said to take weeks to reach their maximum, the chronic cases perhaps months. But in acute spinal cord lesions themselves I think we may recognise at least three different degrees. There is first the acuteness of cases of intra-spinal hæmorrhage—much less common than cerebral hæmorrhage, but still not really infrequent—in which the paralysis reaches its maximum in a few minutes; these might be called cases of sudden paralysis. Then there are the cases of children who, it may be, are put to bed at night apparently quite well, and in the morning are found to be completely paralysed in some limb or limbs; this paralysis, which is a matter of hours, might be called rapid. Lastly, there are the cases in adults in which the paralysis

is not completely developed until several days after the illness has commenced. These, I suggest, might be called cases of deliberate paralysis.

This patient, then, was admitted into hospital on October 21st last, under the care of my colleague, Dr. Chappel, with the history that eight days previous on going down stairs in the morning she felt her left leg weak, and was unable to do her work. She was seen by a highly capable practitioner, who directed that she should be kept in bed. She needed to be helped up stairs by two persons. On this day her temperature was found to be 101° F. She had, however, been feverish for a few days before the weakness was first noticed, and had been ailing for a week before, during which time she had been much constipated. On the following day her temperature was down to 99° F. Two days later, when she tried to get up out of bed, she fell, and had to be helped back to bed again. Before this she had been able to lift her leg off the bed with some difficulty, but after, she became unable to do so. For a few days before admission there had been some difficulty with micturition in the direction of retention; urine was passed at long intervals, when a large quantity was voided. There was never any incontinence. She had never any pain or numbness, and had never shown any tendency to bed-sores.

Before the present illness she had been quite free from serious illness, and there was nothing of importance in the family history.

On admission she was found to be in quite fair, almost good, general health, but on looking at the lower limbs there was obviously flaccid paralysis of the whole of that of the left side. There was, however, then no obvious wasting, and there was no sensory loss and no especial coldness of the paralysed limb. The left knee-jerk was absent, the right was brisk. On the following day a catheter was passed and forty-six ounces of urine were withdrawn. By the end of the next week there had been some improvement in the paralysis; she could dorsiflex the ankle fairly well, move her toes and slightly flex the knee, but most of the thigh-muscles, with the lower back-muscles and the lower portion of the left rectus abdominis, were still paralysed. The muscles were now wasted; the limb above the knee was distinctly softer and smaller than the other. Electrical examination showed loss of faradic excitability in the muscles above the knee, with qualitative changes to galvanism, ACC being equal to, or greater than, KCC.

The essential points in this case from a diagnostic point of view are a flaccid paralysis coming on with pyrexia, and wasting with reaction of degeneration in the paralysed muscles, but without anæsthesia or persistent sphincter trouble; and these can only point to an anterior poliomyelitis. A flaccid paralysis of abrupt onset in children is nearly always an anterior poliomyelitis. A spastic paralysis of the lower limbs coming on more or less gradually is usually a compression paraplegia due to spinal caries. With what might this adult poliomyelitis be confused? It has probably been most often in the past confused with a multiple neuritis, which, indeed, has made it appear less rare than it really is. In toxic multiple or peripheral neuritis, as from alcohol, there is usually much pain and muscular tenderness, glove and stocking anæsthesia, with dropped feet and dropped wrist it may be, and there is usually no pyrexia, and above all it is gradual in development, taking generally weeks to reach its maximum. Of course in certain groups of cases, as after diphtheria or lead poisoning, there may be no anæsthesia or pain, but there are other signs clearly pointing to the nature of the case, as palatal paralysis, a blue line on the gums, and so on, and the history is again quite different. Acute myelitis might also be confused, but here, also, there is anæsthesia; the paralysis below the level of the lesion is eventually spastic, there is usually sphincter trouble, and bed-sores tend to form. Intra-spinal hæmorrhage is a third condition apt to be confused, but here the onset is absolutely sudden, there is no antecedent pyrexia, sensation is usually affected, frequently in a dissociated way, perhaps with tactile anæsthesia on one side and pain and thermal anæsthesia on the other; the bladder and rectum are frequently affected with, sooner or later, signs of lateral slerosis below the level of the lesion. The cause of anterior poliomyelitis is usually a toxæmia, and its result is an interstitial inflammation along the course of the anterior spinal arteries. The way in which the infantile differs from the adult is, in addition to the preferential age, that in the adult the febrile disturbance has lasted some days before the onset of the paralysis, and its distribution is usually more widespread than in children.

What about the prognosis in these cases? As

regards life it is quite favourable; as regards recovery it is unfavourable, but a considerable degree of improvement may quite justifiably be looked for.

As regards treatment, during the early stage the aim is to prevent extension of the process; the patient should accordingly be kept in bed, preferably either lying on one side or prone, so that the spinal cord shall not be more dependent than is necessary. Careful regulation of the bowels is indicated, especially with the object of removing sources of auto-intoxication; for this end calomel and intestinal antiseptics may be employed. The skin should be kept acting also to counteract the toxic factor. In the way of drugs there is no specific; perhaps sodium salicylate has been most in favour. Later in the case, with the object of promoting improvement, the aim is to develop the non-atrophied muscles or parts of muscles, remembering the very wide central representation of the individual groups of muscles. Electricity and massage have seemed in my experience of great help in some cases and of some help in many. Of course to help the atrophied muscles galvanism must be employed, inasmuch as faradism produces no response. These means may be commenced after the third or fourth week, and should be persevered with for at least a year. It is important to prevent contractures by any and every means, especially flexor contracture of the lower limbs. In the way of medicines potassium iodide is perhaps as useful as any.

After a term of, say, years it often becomes necessary to consider how to improve the limb or limbs for locomotion, and even for wage-earning in the poorer patients. Exercises should be begun early and persevered with, the object being to keep the undamaged muscles and muscle-fibres at their highest functional potentiality. Then tenotomy, the transplantation of tendon to muscle, tendon, or bone and other orthopædic devices often result in greatly increased motor activity.

Here in passing I may show you a case of infantile paralysis, of acute anterior poliomyelitis in a child, with which you may compare the adult form. As you see, it affects almost the whole of the right upper limb. It came on with fever in one night. The limb, as you see, is wasted, very flaccid, actually flail-like, and it is, and is accustomed to be, the mother says, distinctly colder than the corresponding limb; it is also bluer. In relation to this point, which is of some diagnostic importance, we remember that vaso-motor centres are represented in the spinal grey matter as well as in the medulla and cortex cerebri.

The great diagnostic point is, however, the reaction of degeneration in the wasted muscles; unless this were clear this disease should not be diagnosed. Taking any case of muscular atrophy or paralysis, if there is no reaction to faradism either the trophic influence of the anterior horns is cut off from the muscles—whether by actual destruction of the anterior horns or by inflammation or other lesion of the peripheral motor nerve-trunks or nerve-terminations — or there is no real physiological muscle left in the apparent anatomical muscle. If there is no muscle substance left no reaction to galvanism will be obtained; if a reaction to galvanism is obtained there is certainly muscle present; if so. and a reaction to faradism is not obtained, then probably the muscle will be more excitable by the positive pole —the anode, than, as in health, by the negative—the kathode, and instead of giving a brisk twitch as in health it will give a slow worm-like contraction, that is to say, the reaction of degeneration is present. A useful practical point in the infantile cases is to know how to get some help from electrical testing in a sensitive, easily frightened little child, and this can often be accomplished sufficiently well for diagnostic purposes by first applying the electrodes of a stationary faradic battery and then moving the Neef's hammer to and fro with one's own finger so as to elicit single muscular twitches, but not a tetanus, in the control healthy muscle, and failing to obtain any twitch at all in the paralysed muscle. This is much less alarming for the child.

The second case is one of myopathy, This patient, a cycle-maker by occupation, is 24 years of age, and as he lies in bed it is obvious from the pouting lips, and transverse smile when he is amused, and from the general appearance of lack of muscle tone in the lower face that he is suffering from some muscle weakness.

His history is that in November, 1899, he complained of difficulty in keeping his head up straight; it tended to fall backwards, or especially forwards.

Later he began to notice that his walking was unnatural; he threw his feet forwards and brought them to the ground with a stamp; limping first occurred on the left side. In 1901 he noticed that his left upper arm was getting thinner; in the course of months the other upper arm and the shoulder muscles began to waste, then practically all the muscles in the lower limbs, and even slightly the muscles of the forearms. Quite recently he has noticed weakness of the muscles of the small of the back and of the hands. He never noticed any weakness of the muscles of the face until he first came to hospital and it was pointed out to him. All along his general health has been excellent. For several years he acted as a chorister, but latterly the higher notes of his baritone voice have been impossible for him. Except for an occasional attack of bronchitis up to the age of fifteen, whooping-cough in childhood and rather troublesome boils, he has had nothing to complain of in his previous health. There is nothing of importance in his family history, and he is quite certain there has been no instance of muscular atrophy among his relatives.

Now on first looking at the man, what especially strikes one is the weakness of the face muscles, the wasting of the shoulder muscles, and above all, the thinness of the upper arm as compared with the well-developed forearm muscles; it is an obviously facio-scapulo (shoulder) humeral distribution. But on looking at him more scrutinisingly one sees that sitting or standing the head falls forward with the chin on the chest, and that the cervical spine shows immediately below the skin as a rounded column bent forwards. The neck muscles are almost completely atrophied. There is considerable lordosis seen when he stands up, but he cannot rise up unaided from the sitting posture, and from the recumbent posture only by the process of climbing up his legs. The orbiculares of the eyes and mouth are very weak; he cannot whistle. The scapulæ are greatly winged. The deltoids, especially in their upper portion, are greatly atrophied. The upper arms seem literally but skin and bone, and the same applies to the thighs. He walks with a very waddling gait. He cannot flex his elbows; when he wants to button his coat, he swings his hand to and fro until it catches on. The back muscles and pectorals are practically gone. Although the peronei and anterior tibials are very weak and small, the calf muscles are of fair size, although very weak; at one stage they seemed hypertrophied, and even now the supra- and infra-spinati are bulky, hard, and lumpy—in a condition of pseudo-hypertrophy in short. Measurements taken a while ago showed the circumference of the arm over what should be the greatest prominence of the biceps about half as much as the maximum circumference of the forearm. There are no sensory changes. The knee-jerks are absent, but the extensors of the knee give no reaction to either faradism or galvanism, and the same applies to the other completely atrophied muscles. The partially atrophied muscles give a reaction to both currents diminished in quantity corresponding with the degree of atrophy, but not altered qualitatively, and this is the characteristic electrical phenomenon in myopathies.

I have already expressed the opinion that this case is one of myopathy. How does a myopathy differ from a myelopathy? As compared with acute anterior poliomyelitis, for example, the development is very slow, taking years. In the other the mischief is done in a few hours, and although there may be improvement there is no downward progress. Both from this and from the more chronic myelopathies, as progressive muscular atrophy, it differs in exhibiting only quantitative electrical changes, in its starting place, and in other points that need not detain us now. The outstanding diagnostic features of this particular type of case are the progressive wasting, the merely quantitative electrical alterations, the loss of the deep reflexes only when the wasting is about absolute, and the atrophy of the face, shoulder, and upper arm muscles before and beyond all other.

What are the main types of myopathy? The standard type, if one may call it so, is the well-known pseudo-hypertrophic paralysis, beginning in early childhood, with fibrous overgrowth or pseudo hypertrophy in the calf muscles, the infra-spinati and some other muscles, in the early atrophy of the latissimus dorsi and the lower half of the pectorals, comparatively useless muscles as will appear to you, and in being transmitted from generation to generation through the mothers, who are not themselves affected, to the sons. Then there is the Laudouzy-Déjerine type, as in our case, beginning as a rule between five and seven,

but often not until somewhat later, usually in the face, and appearing, it may be, in several generations of the same family, but not transmitted through the mothers alone. The last type I shall mention is Erb's juvenile form, beginning usually between ten and twenty, commencing in the shoulders and arms and having a distinct heredity. This is practically the facio-scapulo-humeral form with the face escaping. It may indeed be called a transition form between the first and second types. Are there any close resemblances between the first and last types? Yes, for in our patient there is distinct pseudo-hypertrophy of the infraspinati and calf muscles. Indeed, it may be said that an academical distinction between the types cannot be strictly maintained. As to the ætiology of the condition, we must assume by way of explanation, although it is hardly a real one, that there is an inherited defect in vitality of the muscles, that muscular decay sets in much earlier in these patients than somatic decay. As to the influence of possibly toxic factors this must not now be discussed, but I hope to refer to it in a few days in connection with the hereditary peroneal type of muscular atrophy, which, however, is not really a myopathy. As to prognosis, the tendency of myopathy *per se* is not towards death but intercurrent visceral inflammations are very fatal. As to treatment, the great thing is probably the employment of suitable exercises and games, not with the hope of cure, but of retarding the deterioration. Various kinds of apparatus have been employed to obviate contractures, and when these have occurred, skilfully directed tenotomies in the hands of orthopædic surgeons have added years of useful life in many cases. Our patient is having strychnine injected hypodermically, five minims of the liquor twice a day, but this is, in his case, but little more than a counsel of despair.

December 14th, 1908.

We have received from Messrs. J. & A. Churchill a copy of a 'Dictionary of Medical Treatment,' by Arthur Latham, M.D., F.R.C.P., Physician and Lecturer on Medicine at St. George's Hospital, and Senior Assistant Physician to the Brompton Hospital for Consumption and Diseases of the Chest. The idea of his dictionary is to give a definite course of treatment for any disease commonly seen in medical as distinct from surgical or special practice, and Dr. Latham has succeeded in giving in a succinct and practical form a clear-cut and definite idea of the usual procedure in the majority of cases of any particular disease. The plan of the book is admirably adapted to enable Dr. Latham to sketch out the general methods of treatment to be adopted, whilst at the same time care has been taken by the author to give sufficient detail and precise accounts of remedies where desirable. A very good example of modern up-to-date teaching of the best sort is presented in Dr. Latham's remarks on the method of administration in vaccine therapy—a subject in which he is so well qualified to teach. He says, very truly, that until recently vaccines were always given by means of a hypodermic injection, but it is now established that tuberculin, staphylococci, streptococci, and other vaccines can be given with good results when administered by the mouth on an empty stomach or by the rectum. The administration by hypodermic injection causes a longer preliminary negative phase as a rule than administration by the mouth; consequently, when vaccines are given by the hypodermic method the interval between the doses must as a rule be longer and the dose itself somewhat smaller than when the oral method is employed. If the hypodermic method is employed it is necessary to use strict aseptic precautions, the injections being made into the subcutaneous tissue between the shoulder-blades or in the flanks. When the oral method of administration is employed, the vaccine, suspended in 10 c.c. of normal saline solution, should be given, together with 2 oz. of milk, on an empty stomach some two and a half to three hours after food. Vaccines may be administered with 1 oz. of milk by the rectum by means of a soft catheter and funnel, the patient resting for twenty minutes after each dose. It may be taken as an absolute rule that (1) a dose of any vaccine which is followed by fever and an increase in the severity of the symptoms is too large; (2) better results are obtained from vaccines made from the patient's own micro-organisms than are obtained from stock vaccines; (3) treatment should be continued for some time after all symptoms have gone, to diminish any tendency to relapse.

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A CASE OF LARGE WHITE KIDNEY.

A Clinical Lecture delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P., Physician to the Hospital.

GENTLEMEN,—My lecture to-day is upon two cases of renal dropsy which closely resemble one another, one of whom came in this year and the other two years ago. The case which occurred this year was that of a girl, A. A—, æt. 16 years. She was admitted to Mary Ward on July 15th of this year with extreme œdema all over the body. The history she gave was that she was perfectly well, as far as she knew, until November in last year, and one day she woke up and found her face swelled. The legs swelled at the same time. She went into the London Hospital, where she was two months, and improved, but she never completely recovered. When she was discharged about the end of January she was still very œdematous. She remained in the same condition until the last week in June, and then the œdema increased gradually, and after three weeks of that she came into this hospital on July 15th. She could give no history whatever of any attack of nephritis, and at no time that she could recollect had she had any of those symptoms which we call renal; she had had no pain in the back, such as one finds in congestion or inflammation of the kidneys; there had been no particular scantiness of urine, and she had never noticed any alteration in the colour of her urine, such as one sees when the urine contains blood. Nor had she ever had any cerebral symptoms such as you get with kidney disease, of which symptoms the earliest are headache and sickness. So that her disease appeared to have come on without any acute onset of any kind. Further, she said she had had scarlet fever nine years ago, which we always inquire

after, because it is a well-known antecedent of renal disease. But we could not find that the scarlet fever of nine years ago had left any sequelæ, and she had not had any swelling at that time. Her only other illness occurred two years ago with otorrhœa. She was enormously œdematous when she came to us. There was much swelling all over; legs, arms, and trunk. We could not feel the apex of her heart, nor could we percuss it out owing to the great œdema. The heart-sounds had no murmur, but the beats were what is sometimes called "tic-tac," like the pendulum of a clock, the two sounds being almost exactly equidistant from one another. That means, I always think, a weak, slow contraction of the ventricles, so that the aortic pressure does not rise as quickly and shut the valves as quickly as in an ordinary healthy person. The lungs were practically natural, which was rather curious considering the immense amount of œdema which was present elsewhere. There was some ascites—some fluid in the peritoneum. The urine was at first of considerable amount: in the first 24 hours we measured 68 ounces, which is about 2 litres. It was, however, of low specific gravity, about 1005, and contained very little urea, the amount for the 24 hours being 5 grammes. It contained a fair though not a full proportion of chlorides—2½ grammes of chloride in 24 hours. I always pay great attention to the chlorides in renal cases, and I think the amount of chlorides excreted is almost a better guide to prognosis than is the amount of urea. But it is not always a certain guide, and in this particular instance which I am dealing with it was not a certain guide. In the eyes there were none of those appearances known as retinitis albuminurica. There was a slight haziness of the retina, I suppose a sort of œdema, but none of the white patches or of the hæmorrhages which are characteristic of retinitis albuminurica. You will remember she was admitted on July 15th, and on August 4th she began to have a little sickness and headache, which are always ominous signs in nephritis. On August 10th she had, first of all, a pain in, and then a discharge from, the ear, a regular otitis media, with perforation of the tympanum. On August 14th the throat became sore, and on the 17th we found a great patch of membrane, which had first occupied one tonsil, and then spread right across the uvula and to the other tonsil, exactly like what would happen in a very bad case of diphtheria. But it was not diphtheria, and when the membrane was examined it was found to contain a fusiform bacillus and a spirilla. Therefore it was a membranous inflammation belonging to that form of inflammation of the fauces which is known as Vincent's angina. It is a specific form, which I had never seen before. It is an uncommon form. That recovered after three weeks, under local treatment with antiseptics. But the otorrhœa went on, and eventually she got signs of inflammation in the mastoid process, and so we had to have the mastoid process opened by Mr. C. E. West, and shortly after that she gradually sank and died later on from exhaustion.

We had a case in the hospital almost exactly similar to that two years ago, the patient being a girl, æt. 19 years, Amy M—. She was admitted in March to Mary Ward with almost exactly the same history: universal œdema, albuminuria to a great degree, no history of any acute attack, and in her case no history of scarlatina either. She went on almost exactly in the same way, but she did improve at one time, so much so that we were able to get her down to the Convalescent Home. But she had a fresh relapse there, and came back here and died on May 23rd. In her case, too, the urine was below the usual level, though at times it reached quite a large amount. Until quite the end the urea was passed to the same amount as in the first case, namely, 4 or 5 grammes *per diem*, which is very small, and chlorides in fair proportion were excreted. There was no sign of arterial disease or of any real rise of blood-pressure, and there was no albuminuric retinitis. In her case, too, there was no uræmia, that is to say, there were no cerebral symptoms, fits, or twitching or anything of that kind, and the only signs of what we call uræmic poisoning up to the last day of her life were slight headache and occasional vomiting. Then she became half comatose and half exhausted, and died on May 23rd.

Those two cases are examples of a particular group that is quite well recognised, and both, you will notice, are almost exactly alike in their clinical symptoms—an insidious onset, great œdema, much albuminuria, no retinitis, no rise of blood-pressure, and no uræmia. Each also presented almost the same post-mortem appearances. When we came to do

the post-mortem examination on those two cases the kidneys were found to be in each very large, eight or nine ounces apiece, whereas that ought to be about the total for the two in the case of a girl of that age. They were quite smooth, the capsules stripping off with perfect ease. The outside surface was quite pale, and when we came to cut the section it was of a very remarkable appearance. The cortex was yellowish-white, and came down on either side of a pyramid that was a rich purple. The contrast between the two tissues is very remarkable. When you come to examine microscopically you find that cases like that are distinguished from every other case of renal disease in this: that almost the entire stress of the disease is upon the epithelial tissue. They are not all the same: these two cases were not exactly the same. Still, the stress of the condition is on the epithelial tissues; in that they both agree. I do not believe there is any case of renal disease in which one tissue alone is affected, nor is that so in these cases. In both there was a slight affection of interstitial tissue—quite slight. The arteries are hardly at all affected in either, as you would expect in so young a person. But the tubes in each case were choked full of cells and granular stuff and hyaline plugs of what we find in the urine as albumen, and the glomerular capsules also contained a lot of exudation and proliferated cells from the capsule. And that is the characteristic of these cases, that it is a disease almost entirely of the epithelial and glomerular tissue. The other organs of the body are affected to a certain extent too. The heart in both these cases was rather large, about 13 ounces, which, of course, is large for a girl. In both it was pale, and in one of them, which I examined microscopically, the cardiac muscle was fatty. In both the liver was large, and in one of them which I examined the liver was also fatty. These kidneys have much fat in them, and in one which I examined for that point there was a great deal of fat in the tissues. It is therefore a parenchymatous nephritis, which degenerates in a fatty direction.

What can we do in the way of treatment in such a case as that? Really we can do but little. You know, as well as I do, that we have not yet discovered what kills people who have nephritis. All we can do is to do what we can to restore the secretion of urine. At least we think we are on the right lines if we can manage to do that. And in any case of dropsy, especially, you try at once to drain the patient through some of the natural secretions. I am not at all inclined to drain patients through their bowels by giving them diarrhœa. By that means you are very apt to start an intractable diarrhœa which leaves the patient worse than you found him. And I do not go beyond keeping the bowels slightly open. But of course one naturally tries to increase the quantity of urine excreted. And what are the diuretics at our disposal? They can be divided into three groups. The first includes the neutral salts, citrate of potash, bicarbonate of soda, acetate of potash, and so on. The second group is the digitalis group, which comprises digitalis, strophanthus, convallaria, squill, and one or two others. And the third group is the alkaloids of tea, coffee and cocoa. Those three groups of diuretics act in different ways. They have all three been experimented upon by pharmacologists, and we can safely say we know, roughly speaking, how they act. When you inject neutral salts into the circulation, by the law of osmosis they attract into the blood-vessels the less concentrated fluid from the tissues, so that the injection of salts into the circulation is followed by an inrush of water from the other tissues of the body. And until the equilibrium is established by the outflowing or dialysis of some of the salt there is for the time a greatly increased pressure in the blood-vessels. The result of that is an increased secretion of urine, and that has been repeatedly followed experimentally. That acts well on healthy animals, and it acts generally to some extent upon diseased men. Secondly there is the digitalis group, and that acts in a different way. It is remarkable that in healthy animals it cannot be relied upon as a diuretic. It is sometimes quite impossible to increase the urine by the injection of digitalis in healthy animals. It is not a good experimental diuretic, but in man it is quite well known to be very efficacious sometimes, and the reason is not very difficult to see. In a great many cases scanty urine in men depends on venous stasis due to heart failure. There is a back-pressure on the kidney, and in consequence the flow of blood is greatly delayed. That we know to be a cause of scanty secretion. Digitalis strengthens the heart, relieves this back-pressure, increases, of course, the rapidity of the circulation, and instantly increases the secretion of urine. That is a very

well known fact in man, and the reason digitalis does not act in dogs as a diuretic is easy to understand. It is because the animals used are not suffering from heart failure. The third group is comprised of the alkaloids of tea, coffee and cocoa, caffeine and theobromine. They are almost exactly the same. Caffeine is a xanthin, to which three methyl groups have been added, and theobromine is a xanthin to which two methyl groups have been added. Theobromine is derived from cocoa, and it appears that every new manufacturer produces a new specimen of it and gives it a new name. Theobromine, theophilline, and theocin are, for instance, all advertised as diuretics. But as a matter of fact they are all the same; they are differently labelled because the different labels enable them to sell better. There are also other productions derived from them, such as diuretin, which you see constantly employed in the wards, and, indeed, I generally use it myself. It is theobromine sodium salicylate. Theocin sodium acetate is an analogous salt. Agurin is, I believe, exactly the same thing. Those substances are practically theobromine and nothing else, and their action is the same in all. So far as we can tell from experiments on animals, they have a definite action upon the kidneys. They differ, that is to say, from both the other groups of diuretics in that they are specific renal stimulants. Whether they act on the nerves of the kidney or whether they act on the renal cells seems a little doubtful. But, as you can imagine, a renal stimulant is not of very much use if the specific renal tissue is altogether destroyed. And that is practically what was the case in both these girls, and in all such cases of large white or fatty kidney. We treated this girl by the following different steps so as, if possible, to increase her urine. We began with neutral salts on July 17th, and gave them for a week, but with no effect. On July 24th we began with digitalis, and I gave her half an ounce of infusion of digitalis. There are several preparations: the infusion, the tincture, and the powdered leaf. A very good preparation is that pill which we have in our pharmacopœia: pil. hydrargyri et digitalis co., a grain of mercury, a grain of powdered digitalis and a grain of squill in each pill. We gave that for a week, but it did no good. And in giving digitalis do not be afraid to give enough. Some people give minute doses of digitalis and expect it to have an effect. Very often you have to give large doses, for instance 20m of the tincture, which is above the pharmacopœial dose, and I am not afraid to give that for two or three days at a time to that extent. On the following week, from July 31st, we tried the other group of diuretics and gave diuretin, but still with no good result. On August 5th we tried another member of the group, theocin sodium acetate, but without effect; and then we combined it with the pil. hydrarg. digitalis co. twice a day. As there was no good result from that we gave on August 6th, as the urine was foul, a urinary antiseptic, urotropin. On August 13th we tried something quite new, after consultation with Dr. Samuel West, and gave her a watery extract of sheep's kidney every day for a few days. But this also was useless. Meanwhile we had been trying other measures. When she first came in I cut off all salt from the diet: her food was cooked without salt, her bread was made without salt, and her butter was made without salt. That is sometimes a very good treatment for dropsy. The urine always contains a certain amount of salt, and if you put no more salt into the body the urine sometimes will drain the salt, and the water with it, out of the rest of the body. But in a little while her appetite absolutely failed. She could not go on with her food while there was no salt in it. And that is the case with some of these patients. Even while it was continued we could not trace any good from it. For the first three days we tried the effect of hot-air baths. You know the apparatus in the ward: an air receiver with a long spout and a lamp under it; the lamp is lighted and the air goes up the spout and under the bed-clothes into the bed. We tried also pilocarpine, and that produced profuse sweating. It is not a drug which is commonly used in uræmia, and many people object to using it because it in itself produces convulsions in animals. But I have used it often in cases of uræmia in the hope of relieving in some way the retention of fluids in the body, and possibly getting rid of something besides the water, and always without any bad effect. In this case the effect was *nil*. On this girl I tried half a dozen leeches to each of the kidneys, but without any effect. And one could see the reason for that afterwards: that there was no congestion in these kidneys, and therefore such a local relief of congestion as can be produced by leeching had no effect. But after this the throat and ear started to be bad, and she was too ill for us to do anything more than try to keep her alive as best we could. We could not use local or general remedies to any great extent, and she died, as most of these cases die, apparently not of uræmia, that is to say, not of any direct poisoning from the kidneys, however that may be brought about, but partly by exhaustion and partly, as so many of these cases die, by a slight secondary inflammation, sometimes in the heart, sometimes in the lungs, and in her case in the throat and ear.

December 21st, 1908.

WITH MR. BLAND-SUTTON AT MIDDLESEX HOSPITAL.

II.

GENTLEMEN,—During the last few weeks I have had quite a run of breast cases, as you can see by this assortment of specimens on the table, and I have had them preserved practically as they were removed, so that you might see many of the chief features which they showed, as near as possible, during life. When you come to work in the out-patient room, one of the things which you will notice most is that cancer of the female breast is one of the commonest diseases which will come under your observation. Indeed, the breast, with the possible exception of the stomach, may be regarded as the commonest situation in the body in which cancer occurs. And although surgeons have been looking at tumours of the breast since the time of Hippocrates, and even before that, yet at present there is absolutely no unequivocal sign by which you can tell that a patient has cancer of the breast. When the disease is well developed there is, as a rule, no difficulty, but in the early and curative stages you will find that the cleverest, wisest and most experienced men who look at the breast will often be in considerable doubt as to whether the lump which they feel in it is really cancer or not. One of the most extraordinary facts which will strike you when you look at these breasts is this: that in some cases the knot in the breast, which is called cancer, is sometimes, when cut into, scarcely larger than a cherry, while in another case it will form a tumour as big as your fist. Examples of that you will see on the table before you. Here is a breast with a definite tumour in it, which is about as large as a chestnut, while here is another with practically no tumour in it at all, but an indefinite hardness over an area a centimetre square. Here is a mass in the breast as large as a tennis ball, and here is another with a tumour as large as a Jaffa orange. Before the days of pathological histology, before coal-tar colours were discovered, those who studied pathological histology were put about as to how to differentiate between these tumours; in fact, there was really no such thing as pathological histology. But nowadays we have learnt to cut fine sections of these tumours and stain them with hæmatoxylin or some coal-tar preparation, such as an aniline dye. By such means we get some of these beautiful slides illustrating disease, some of which are really works of art. This branch of histological chemistry has enabled us to tell the differences between various tumours. In the days before these methods were known a tumour in the breast was called a scirrhus or stone cancer if it was small and hard; when it was large and soft and massive like the one I show you it was called an encephaloid or brain-like cancer, or medullary cancer; and if it contained much jelly-like material it was termed a colloid cancer. But now, by means of the microscope, aided by differential staining, we are able to show that these tumours, although many of them when they are cut across do not look like cancer, are found to be so when they are sectioned and stained. And what is more important in the morbid or histological point of view is that an investigation into the characters of these tumours enables us to classify them, and a much more important thing is that the surgeon can tell, when he knows the minute structure of these tumours, whether he is dealing with an innocent or a malignant tumour. And if he is dealing with a malignant tumour it enables him to make what, in ordinary clinical language, we call a prognosis. Before we deal with these specimens I must ask your attention for a few minutes to the rough anatomy of the breast.

Of course the breast varies in size in different individuals. Fat women have enormous breasts, and, as a rule, thin breasts are found in thin women. Still, sometimes slim women will be found to have large breasts, but a big breast in a thin woman usually consists of a large amount of glandular tissue and a small proportion of fat. The essential element of the breast is the teat, nipple or mamilla, which is of different shape in different people. In some it is below the surface, and is said to be inverted: in others it is small and prominent, and in those who have borne many children and suckled them it is large, thick and fleshy. When you examine the nipple with a magnifying glass you will be able to see small ducts; these run from the nipple into the glandular substance of the breast. The large mass of the breast, which rests on the pectoralis major and serratus magnus muscles, is usually spoken of as

the body of the breast; it contains the epithelial elements which manufacture the milk. The breast is a compound racemose gland, consisting of about twenty lobules, each of which has its own duct, which traverses the nipple and opens at its tip independently of its companions. The ducts terminate in the substance of the breast in what are known as acini. In breasts which have never been functional the epithelium lining it is so closely apposed as to resemble a solid column; in functional breasts the acini possess comparatively large lumina, and the epithelium is tall and columnar; it is, in fact, the epithelium which manufactures the milk. At the base of the nipple each lactiferous duct presents a dilatation or ampulla, known as the sinus.

The point I want you to understand is this: A system of epithelium-lined canals terminating in acini also clothed with epithelium permeates the breast from the tip of the nipple to the utmost confines of the gland, and from any part of these epithelial canals a malignant tumour may arise, and when it develops in the acini of the gland it is spoken of as acinous carcinoma, and is the common kind, but when it develops in the sinuses and ducts near their terminations it is called duct carcinoma. This is an uncommon kind, but it is worth study because it is not so malignant as the acinous form of cancer, and its presence is announced by a very characteristic sign; the patient complains of a free discharge of blood-stained fluid from the nipple, and sometimes the amount of blood-stained fluid is so abundant that it soaks the clothes, and women often seek relief entirely on account of this blood-stained discharge, and not so much on account of the tumour which they find in their breast, or to any annoyance it may cause them.

Although duct cancer is not so malignant as the acinous form, nevertheless it sometimes recurs. When this happens the recurrences take the cystic form; they appear like over-ripe grapes. Sometimes these little cystic bodies are filled with blood-stained fluid, and possess what is called an intra-cystic process; this process resembles often a small unripe (red) blackberry. So the cystic character of this duct carcinoma is preserved in the recurrences as well as in the primary tumour. I have said that acinous carcinoma may arise in any part of the breast, but the common place in the breast in which it arises is underneath the nipple. As soon as a patch of acinous carcinoma grows at the base of the nipple the connective-tissue reaction is established, and the fibrous tissue formed in consequence contracts as it grows and draws in the nipple. And in nine cases out of ten when you look at a cancerous breast, if the carcinoma is in this classical spot, that is, round the base of the nipple, the nipple is found to be retracted, the breast shrunken, and it is smaller than its fellow. The appearance thus presented resembles the crown of a ripe pippin.

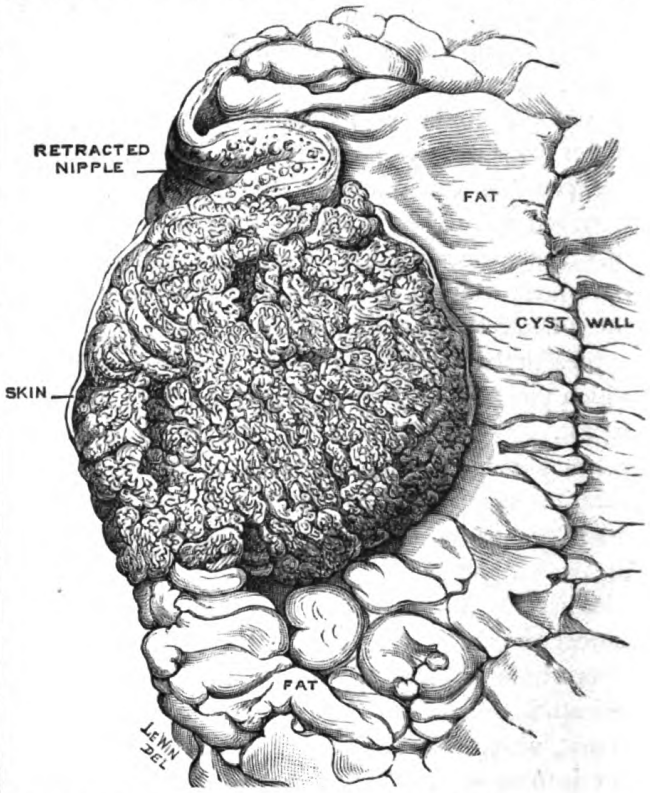

FIG. 1.—A cancerous breast in section showing retraction of the nipple.

Surgeons in examining a breast always look carefully at the nipple to see whether it is retracted or not. I want to say a word or two about this retraction of the nipple, because we have some specimens on the table which illustrate this in a very interesting way. Here is a well-marked case where the nipple is retracted (Fig. 1), and here is one where you might think the nipple is retracted but that is not so. In some cases the nipple, instead of being drawn in and retracted, is inverted. Inversion of the nipple is quite different from retraction. For instance, instead of the nipple standing out it will be drawn in, and it hurts the

patient to pull it out. After the operation is done, if you take a pair of forceps you can draw these nipples and bring them out and make them pro-

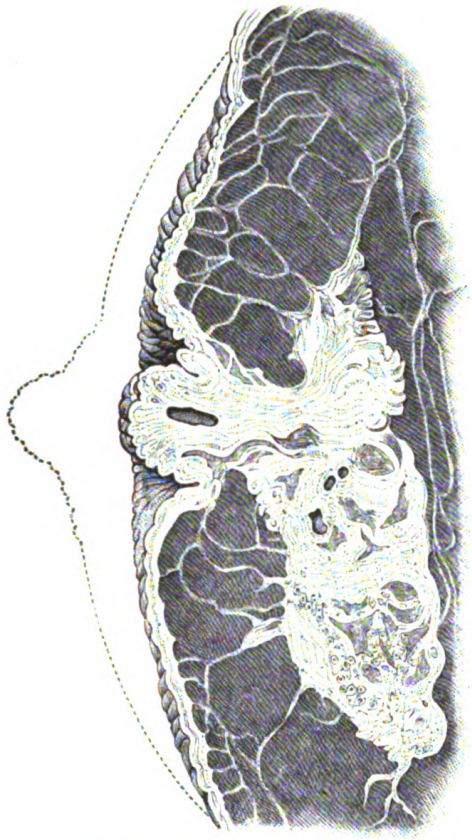

FIG. 2.—A breast with its nipple in section from a case of duct cancer. The nipple is inverted.

minent like a normal nipple. But you cannot do that with a nipple which is retracted in the ordinary way by carcinoma. This form of inverted nipple (Fig. 2) is met with in many individuals, and it is a normal thing, and in others it is inverted when they suffer from duct carcinoma, and sometimes when they suffer merely from cyst in the breast. The most important thing from the clinical standpoint when you are looking at a breast suspected to contain cancer, and you cannot see the nipple standing out prominently, is to make sure whether it is retracted or simply inverted. Among these breasts there is one with a tumour in it as big as a tennis ball and the nipple standing out in a natural manner (Fig. 3). This is one of the most interesting tumours on the table, because we looked at it in the ward when the patient was admitted to the hospital, and we first noticed at once the extreme prominence of the nipple, although that tumour had been growing in the breast, according to her account, for two years. This made us a little suspicious as to whether it really was cancer. As a matter of fact it is not ordinary cancer, but an endothelioma—quite a rare kind. There is another point with regard to the breast which I must draw your attention to, as concerns the origin of cancer. I have already told you, not only may cancers begin in a lactiferous duct, or in its sinus, or in the acini where they are near the nipple, but they will grow in any part of the breast, even in its most outlying parts. And this becomes a matter of some importance, because when you find a lump in the breast close up under the clavicle or near its axillary border, or close to the sternum, you may be doubtful as to whether you are dealing with a tumour of the breast. The bulk of the breast is immediately under the nipple, but if you examine carefully you will find that it does not form a definite circular mass flat on the

FIG. 3.—A breast in section; it contains an endothelioma (perithelioma). The nipple is unaffected.

chest wall, or as you might think from looking at Luschka's drawing which is figured in the text-books on physiology and many text-books of

anatomy. As a matter of fact there is a tail-like process of the breast which extends towards the sternum, and is known as the sternal process. There is another piece which creeps up towards the clavicle, and that is sometimes spoken of as the clavicular process of the mamma or breast. There is another piece which extends into the axilla, and that is spoken of as the axillary process. And to still further complicate the matter, you know that there are very large skin glands in the axilla, and sometimes these skin glands become enlarged and form large masses of glandular tissue, and when women have children these "axillary lumps" sometimes discharge milk. Quite a number of carefully authenticated cases are known. And it is an important thing to remember that carcinoma, the ordinary form of it, may start in these axillary skin lumps. And it is important before you decide that a particular malignant tumour in the axilla might be in an axillary mammary gland to remember there is a tail-like process passing up towards the axilla derived from the main gland. As a matter of fact, cancer may start in this axillary or sternal or clavicular process, not, it is true, so often as underneath the body of the gland, but still in a fair number of cases. And when it does start under the clavicle in that way and you feel it in the early stages you may have some doubt as to its nature. I have heard myself many times grave doubts expressed, as the growth had started so far away from the main body of the breast, as to whether it could really be regarded as a case of mammary carcinoma. There is an example of this on the table. We noticed there was a retracted nipple, and we also noticed that she had a small hard patch under the clavicle. And I expressed a careful opinion about it. She was brought by a lady doctor, and I told her that although I was not sure whether the lump which we felt under the clavicle was really carcinoma or not, it was sufficiently suspicious for me to recommend the patient to submit to removal of the breast, because it was clear that the breast was undergoing secondary changes. She was fifty years of age, and therefore not likely to have any more children, so I strongly advised the patient to be on the safe side and have the breast removed. But as the breast is such an adornment when ladies are at dinners in evening dress they are anxious not to part with them, especially if they have what is termed an "effective bust." But when I see them sitting round a table, each one envying her neighbour, I often think of the lines:

"The rich and poor alike their nakedness display;
The poor, because they must, the rich, because they may."

However, if it comes to a question of their health and future life the question has to be settled. But this woman took my advice and agreed to part with her breast, and when she lay on the table under the influence of chloroform, and I could feel it distinctly, I had no doubt that it was carcinoma. So I removed the whole breast very thoroughly with the pectoral fascia and the axillary fat and the slightly enlarged axillary lymph-glands. The cancer formed a lump as big as a cherry-stone on the tip of the sternal process of the breast. It may puzzle you to believe why a thing so small as that should wreck the patient's life. It was microscoped and found to be typical carcinoma. Another woman had a small knot in an out-of-the-way part of the breast; we examined her in the ward, and felt no doubt in our minds that this patient had a small carcinoma in the breast, but there was no retraction of the nipple. We thought we could feel some axillary glands, and when we came to operate upon them what we thought to be cancer turned out to be a cyst in the breast, formed in the acini of one of these glands and filled with fluid. But as the whole breast was in the condition known as chronic mastitis, in which the bulk of the breast is replaced by fibrous tissue, and with very little glandular material in it at all, we had no hesitation in taking away the breast, because it has been shown over and over again that cancer is much more liable to attack diseased organs than those which are healthy. Conditions of glands which predispose them to attacks of cancer are spoken of as pre-cancerous conditions. And now it has become recognised that although we are absolutely helpless in curing well-marked cases of cancer, even by operation, we are striving more and more to recognise what are the preceding conditions, or pre-cancerous conditions, so that we can deal with them surgically, before they become actually converted into cancer. I show you these two cases together for a good reason: here is a woman who had really cancer in an outlying portion of her breast, and which we thought was a cyst, but we thought it prudent to advise her to allow

us to remove her breast. She had cancer. And here is another one, which we thought for certain had cancer in the breast, and yet it was a cyst and not cancer. Still, in this second case we removed the breast so that it should not become cancerous, because it is a condition of breast which is often found to precede actual cancer. Here is another breast, and an extremely interesting one, because it shows you the way in which cancer begins right on the nipple, at the very end of the ducts. And the story which this patient tells is very instructive. You see there is no nipple at all; it is not retracted, it is not inverted, it is absolutely wanting. The story was that she was in Northumberland Ward three years ago for rheumatism, and I was asked to see her breast because she had some ulceration of the nipple, and I asked her to wait until the physicians cured her rheumatism. If at that time the ulceration still went on she had better come and see me again. That form of ulceration is known as chronic ulceration of the nipple, and it is fairly common in women. It generally gets well, but there are a fair number of cases in which it becomes chronic, and Sir James Paget, who was the greatest authority in his day on cancer and tumours of the breast, pointed out that he had seen a number of these cases of chronic eczema of the nipple go on until those patients had real cancer of the breast, and then had to submit to operation for it. He advocated and practised amputation of the breast when patients came under observation with chronic eczema of the nipple. This particular woman took me literally at my word; the doctors had not cured her of her rheumatism, and she did not come to me. But eventually this breast got so unsatisfactory that she thought she would apply for relief. You see this ulceration has gone on until it has destroyed her nipple. All round the edge of the ulcer there is a small ring of hard tissue, not larger than a finger-ring, yet which if allowed to remain would inevitably destroy the life of that patient. You will naturally say to me, How is it that, although it has been growing for three years, there is so little cancerous material? It is because this cancer has been exposed to the air and has become infected and ulcerated, and as fast as the cancer forms it slowly ulcerates and comes away as a slough, whereas when it lies in the interior of the organ, as in the case which I show you, before it ulcerates it may form a large mass. I could not imagine more striking examples than these. Here is another breast which has gone on and not ulcerated, but is just beginning to involve the skin. And that is a very important feature in all these cases of cancer. As they grow they contract the breast and then involve the skin, and in time that skin gets thin, then sloughs, and leaves a stinking ulcer. Here is a cancer which has been growing for two or three years, and it has grown on the sternal side of the nipple and formed a definite tumour, and produced marked retraction of the nipple. Anybody looking at that, even a dresser who had only been appointed in the out-patients' department for three weeks, would be able to say it was a carcinoma of the breast. Operation does very little good. Here is a lump in the breast which involves the skin, which has retracted the nipple and made the breast smaller than its fellow, and above all it is associated with a cluster of enlarged lymph-glands in the axilla. Anything more certain than that that was a case of carcinoma could not be in the world. It is as plain as snow on the summit of Mont Blanc.

Here is a case of a different kind. This poor woman had had cancer of her breast, and like so many of these patients she kept her secret; she did not like to display it. But when this was fresh and œdematous it stood out as a mass almost as large as her head, and when we took it off a large amount of serum escaped. And here you can see a large growth in the substance of this breast. This is what the older surgeons would have called a medullary cancer. But from some observations which we made on this growth we were sceptical as to whether it is really ordinary cancer; we must have some further examination of it. (This tumour proved to be a sarcoma of the breast.)

I must say a few words about the endothelioma of the breast, which I have already mentioned. The patient was 65, and had a movable smooth lump as big as an orange in the breast associated with enlarged lymph glands; that is an important thing, and she had this plum-coloured tumour, with no ulceration upon it, although it had been present two years. We were certain there were no signs of dissemination; we examined the liver and lungs and other organs. She had a small trace of albumen in the urine, but we gave her the advantage

of operation. When we did this operation I was so certain it was not a common form of cancer that I asked the Surgical Registrar to take a piece of it to the Cancer Investigation Department, so that we might have information as to what it was for this afternoon's lecture. The tumour was not hard like an ordinary acinous cancer, for it appears as a definite tumour in the breast, the centre of which is breaking down. You might think on looking at it that it was a huge gumma in the breast. It is really a perithelioma (Fig. 4), a sub-variety of the tumours known as endotheliomata. Presently I shall ask you to look at the sections under the microscope. An endothelioma is a cell growth in the coats of the artery. This is really a tumour arising from the blood-vessels in the breast, and

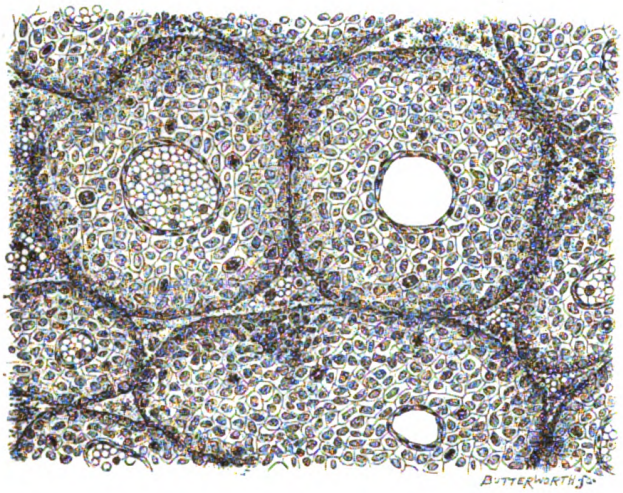

FIG. 4.—The microscopic characters of a perithelioma, showing the cell-mantle around the blood-vessels (after Ziegler).

consists of cylinders of malignant cells formed in and around the coats of the blood-vessels. They are very rare tumours, but an interesting thing about them is this: In the ordinary way, if there had been no such thing as microscopic pathology this tumour would have been cut across and the surgeon would cast it on one side and say, "Oh yes, medullary cancer," and would have taken no further interest in the matter. But now we know it is an endothelioma.

And in regard to these tumours we know that although some of them are moderately malignant, many are benign. In some instances these patients, when they get well, have no recurrences of any kind. So we are able to assure the patient's friends that although the tumour looked ugly and we thought it was cancer the outlook for her is better than we expected it would be.

Now, a thing which will be very interesting to you when you come to study these things in the out-patient department is, that although there are a certain number of cases which come under observation, in which the youngest dresser will have no difficulty in saying it is cancer, yet there are also cases in which, if you have half-a-dozen surgeons to see the case, three will take one side and three another in regard to their opinions as to whether the lump in the breast is malignant or not. However, we are beginning to understand these matters, and the rule which most men are using in regard to tumours in the breast is this: when a woman has a lump in her breast, which has the ordinary physical characters and signs of carcinoma, it is the wisest thing, particularly if the woman is over thirty-five years of age, to advise her to have the breast removed while the disease is in the early stage. Never advise the patient to keep under observation, and meantime smear the breast with belladonna or some other messy and disagreeable pharmacopœial preparation, and "watch" the case. Doctors used to watch six, eight, nine months or a year, and by that time the breast had enlarged and begun to ulcerate, and then when they have arrived at the certainty that it was cancer the disease was quite beyond the reach of helpful surgery. Directly you get a patient who has a suspicious lump in the breast, especially if she is over thirty-five years of age, then, not only for the patient's peace of mind, but also for the welfare of her body, advise her to have the whole breast and the axillary lymph-glands extirpated.

December 21st, 1908.

Electrocution.—The addition of a volt or two to those which inhibit respiration will stop the heart of an animal and cause death. This proceeding contrasts by its gentleness with that used in America for electrocution. With intermittent currents the functions of the superior centres of perception are the first to be abolished and then those of respiration and of the heart. Also no reaction can be produced in the subject, and it is no metaphor to say that one is inducing eternal sleep.—*Medical Electrology*, vol. viii, No.8.

SOME CAUSES OF DEATH DURING ANÆSTHESIA.*

By R. J. PROBYN-WILLIAMS, M.D.,
Instructor in Anæsthetics at the London Hospital.

THE present-day teaching on anæsthesia, and the multiplication of text-books, have made the ordinary uses of anæsthetics so well known that it would be almost improper to occupy your time this afternoon with a consideration of such a question as the choice of the most suitable drug for certain persons, and for certain operations.

But the occurrence of death during anæsthesia is demanding considerable attention, especially as certain coroners are apparently anxious to extend the limits of their usual investigations, and in the next few minutes I should like to discuss one of the aspects of this important subject.

When chloroform and ether began to be employed in every-day practice deaths occurred, and in nearly every instance they were due to an overdose of the drug. But with their still further use a more accurate knowledge of their properties was obtained, and the patient ran less risk of dying in the early stages of the administration. The surgeons gained more confidence, and, as a result, operations of increasing difficulty, lasting for longer periods, and requiring far more care in the administration of the anæsthetic, have gradually become more and more common. Deaths unfortunately still occur, and too frequently, but instead of a fatal syncope during the first few minutes of the inhalation, we hear of many cases in which the patient is kept alive during operations of great severity, and requiring one, two, or more hours for their completion, and finally dying from causes of which the anæsthetic is only one of several which may have contributed to the fatal result.

In my official position as Instructor in Anæsthetics at the London Hospital it is one of my duties to investigate every case in which death occurs in connection with the administration of an anæsthetic; and I can assure you that in many instances it is extremely difficult to determine the exact amount of blame that should be attributed to the effect of the drug employed. I therefore thought that a few minutes might profitably be given to the consideration of some of the causes, other than the direct effect of an anæsthetic, which may result in death during an operation.

All the cases which I quote have either come under my notice at the London Hospital, have been related to me by friends, or have been reported at the meetings of the Society of Anæsthetists.

During the first stages of the induction of anæsthesia fear may be an important factor in causing a catastrophe. Simpson, in his 'Defences of Anæsthesia,' relates several cases of death in the pre-anæsthetic days which occurred from fear.

One gentleman was to be operated upon for a strangulated hernia, and when the groin was being shaved he complained of feeling faint and died. A child with a large abscess in the neck, the walls of which were so thin that no pain could have been caused by the incision, died as it was opened with a lancet.

The most important case may be quoted in Simpson's words:

"After discovering the anæsthetic effects of chloroform in November, 1847, I tested it in a case of tooth-pulling, but required to wait eight days before I had an opportunity of using it in the hospital or elsewhere in any surgical operations. A few days, however, after its discovery, a hernia, which had been strangulated for a few hours, was brought into the infirmary, and Professor Miller thought it a case demanding operative interference, and one on which chloroform should be tried. But I could not be found in time for the purpose of giving it, and the patient was operated on without an anæsthetic. Professor Miller had only proceeded the length of dividing the skin when the patient fainted, and died with the operation unfinished.

"If the chloroform had happened to be used, aud this fatal syncope had occurred while the patient was under its action, the whole career of the new anæsthetic would have been at once arrested."

An interesting case was reported to me last year which occurred in general practice.

A nervous boy, æt. about 9 years, was to have his tonsils and adenoids removed under chloroform. He was placed on the table, and a relative was holding his hand. The mask was held before his

* Delivered before the Hunterian Society.

face, and before any chloroform was poured on it the patient began to breathe rapidly, drumming with his heels on the table, and saying "I'm going." The anæsthetist tried to quiet him and told him that he had as yet had no anæsthetic, but the rapid breathing continued for a few seconds, and the boy died.

Nothing abnormal was found in the chest before the administration, and no post-mortem examination was allowed.

In this instance I cannot see that any cause of death can be ascribed other than extreme fear, and, physiologically speaking, the heart's action was probably arrested by some inhibitory influence of the vagus.

Chloroform certainly cannot be blamed, but what would have been said of it if one or two minims, quite insufficient to procure unconsciousness, had been dropped on the mask? I am confident that some persons at least would then have attributed the death to the sole effect of the anæsthetic.

Of course, when a patient is once rendered unconscious by an anæsthetic he cannot be influenced by fear, but till consciousness has been abolished we must remember that fear can be at least one cause of death; and even if no such disaster result the re-arranging of the table or the mackintoshes, or loud noises made by dropping instruments from the steriliser into dishes, may perturb a nervous patient very considerably during the early stages of the administration.

One of the most common causes of death under anæsthesia is obstruction in the respiratory passages, the fatal issue resulting partly from asphyxia, and partly from the effect of the anæsthetic vapour which is prevented from leaving the lungs.

A careless administrator may allow the tongue to fall back; but there are many other ways in which the respiratory passages may be obstructed. A large thyroid may press on the trachea till it becomes so flattened that respiration is difficult when the patient is conscious, and in the most favourable position. When ether is given to such a patient the mucous membrane becomes congested, and the air-way may become insufficient, and the same result may occur when the head is placed in an unsuitable position.

Vigorous efforts on the part of a surgeon removing an enlarged thyroid or glands of the neck may cause cessation of respiration, either from constriction of the trachea, or possibly, in some cases, through vagus inhibition.

The risk of giving ether to persons suffering from cellulitis of the neck should always be remembered, for in severe cases of this description there is generally some co-existent œdema of the larynx, which may be increased by the effect of ether till it produces asphyxia. Even then, when laryngotomy is performed quickly, the patient's life will generally be saved.

Asphyxia may be caused by the inspiration of vomited matter.

This accident occurs most frequently in those cases of intestinal obstruction in which the vomiting is almost continuous, and in large quantities. By keeping a pillow under one shoulder, however, the vomit may generally be made to run out of the mouth, for it is nearly always fluid.

But this danger may threaten the life of a patient who undergoes an operation so trivial that due preparation has been neglected. The danger of anæsthetising a patient with solid food in his stomach is well illustrated by the following case.

A man, æt. 36 years, was brought to the London Hospital late one night with a lacerated wound of the forearm, which was bleeding freely. It was necessary to operate at once. The man had been drinking heavily, but said that he had had nothing to eat since his dinner in the middle of the day. An attempt was made to anæsthetise him with ether, but as it was not very successful chloroform was substituted. He was under its influence about twenty minutes, and five minutes after the administration was discontinued he began to vomit. His head was turned to the right, and he brought up about a pint of fluid smelling strongly of alcohol. This escaped quite readily from the mouth, but the anæsthetist was not satisfied with the man's colour, so the mouth was opened widely with a gag and a large quantity of potatoes and meat removed from his mouth. The colour still did not improve, and on examination with a finger a mass was felt in the larynx. The head was drawn over the end of the bed, tracheotomy was quickly performed, and a piece of potato removed from the larynx.

The patient was still breathing, but not satisfactorily, so he was turned over on to his face with his head still over the end of the bed, and attempts

were made to remove the obstruction, which was feared to exist low down in the respiratory tract, by thumping the back. Artificial respiration was of no avail, and the patient died.

On post-mortem examination a piece of meat an inch long and a third of an inch thick was found in the right main bronchus. There was a smaller piece in the left main bronchus, together with pieces of potato, etc.

The operation was one of urgency, and the man stated that he had had no solid food within twelve hours of the administration of the anæsthetic. It may not have been true, but it was all that the anæsthetist had to go upon. The vomiting could not be prevented, though it was postponed till the surgical procedures were completed, and I believe that the anæsthetist and the house-surgeon lost no time in their attempts to remove the vomited matter.

The following case illustrates an unusual way in which asphyxia may be produced.

A man, æt. 52 years, was suffering from a malignant stricture of the œsophagus, and was anæsthetised with chloroform for an examination with bougies. He took the anæsthetic badly, strained a good deal, and became blue.

It was decided to perform gastrostomy, and the next day chloroform was again administered. The patient struggled, and then became very blue, and it was obvious that there was some obstruction to respiration. Tracheotomy was performed, but it gave no relief, and it seemed that there was some obstruction lower down in the respiratory tract, as with artificial respiratory movements hardly any air could be made to pass backwards and forwards.

The man died, and at the post-mortem examination it was found that a mass of the growth producing the stricture of the œsophagus had become detached, and was blocking the bronchi.

A patient suffering from an abscess of the lung, or from an empyema which has ruptured into the lung, has been asphyxiated when he has been placed on the operating table with the affected side uppermost. The pus passes along the affected bronchus to the bifurcation, and then enters the bronchus of the healthy side, and places the healthy lung out of action.

When the anæsthesia is very light the pus may be coughed out, and this is a strong argument against allowing a deep state of anæsthesia in these cases, for if it is allowed to become so deep that the fluid is not coughed out, the patient is practically drowned.

The position of the patient is as important as the depth of the anæsthesia. It should be regarded as a fixed rule that when there is pus in one side of the chest that side must always be dependent during an operation, partly to enable the lung that is carrying on the process of respiration to be unhampered in its work, and also to prevent any pus entering the bronchus of the sound side.

Several ways of maintaining the patient in this position had been adopted. One is to allow the pelvis to rest on one table, while the head and shoulders rest on another table, or are supported by an assistant, the surgeon being between the two. Another way is to make use of Carter Braine's arm-rest.

A potent cause of death is direct interference with the proper action of the heart. This has arisen in several ways.

Two patients died at the London Hospital within six months from the unusual accident of cardiac embolism.

The first, S. C—, æt. 35 years, was anæsthetised with ether, and her right breast removed for a fungating sarcoma. The examination of the chest before the administration did not reveal anything abnormal. The anæsthetic was taken fairly well, but just as the tumour had been removed the colour became very bad. No pulse could now be detected, but respiration continued for about a minute longer.

On post-mortem examination a clot was found in the right ventricle. It consisted of a thick portion about 1 in. long and $\frac{1}{2}$ in. in diameter, which was irregularly cylindrical, while projecting from one extremity were three longer portions, thinner, and from $\frac{1}{8}$ to $\frac{1}{4}$ in. in diameter, and from 2 to 4 in. long. This clot had evidently been formed in some vein, and the branches of the left internal iliac vein were found to be partially thrombosed, and the clot in the heart was found to correspond exactly with the part of the vein which was empty.

There was no doubt in the mind of the pathologist who made the examination that the clot in the heart had been formed in the left internal iliac vein and its branches, that it had become detached, and, being swept into the heart with the circulating blood, had caused the death of the patient.

The second patient, A. K—, æt. 52 years, was anæsthetised with A.C.E. mixture, and ovariotomy was performed. A week later a tender swelling appeared in the right parotid region. This was incised under nitrous oxide, but the swelling increased in size and tenderness, and the temperature rose to 101° F. Chloroform was given, and the swelling opened and drained.

The anæsthetic was well taken, and the patient was being taken from the theatre to the ward when the sister noticed that she suddenly became blue and stopped breathing.

On post-mortem examination two clots were found in the right ventricle, one small, but the other ½ in. thick and 4 or 5 in. long, and dividing at one end into five distinct branches. The pathologist considered that this was a case similar to the one just quoted, but in this instance the empty vein was not discovered.

Both these patients were in a septic state, and therefore in a favourable condition for thrombosis to occur.

In the second case it is probable that the clot was displaced during the first efforts to vomit; but the first patient was well under the influence of ether at the time that death took place, and it is not easy to discover why this clot became dislodged.

The entry of air into a vein has caused death during anæsthesia, and an instance of this was reported from Westminster Hospital in the spring of this year. A large mass of glands had been removed from the neck; a vein was punctured during the closure of the wound, and air entered with a fatal result.

A case, which, as far as I know, is unique, occurred at University College Hospital last year, and was reported to the Society of Anæsthetists. A carman, æt. 44 years, while eating his supper, was seized with intense pain in the epigastrium and left hypochondriac regions. He jumped up and tried to cross the room, but fell down unconscious, and remained so for half an hour. He regained consciousness, but still complained of acute pain in the upper part of the abdomen, so he was taken to the hospital. His temperature was 95° F., and pulse 100 in rate, regular, and of fairly good tension. The respirations were shallow and 40 to the minute. His complexion was described as earthy, and his lips had a bluish tinge. Nothing abnormal was discovered in the chest by percussion or auscultation. The heart's impulse was described as heaving in character, and the sounds were weak but otherwise normal. The area of cardiac dulness was not increased.

After a consideration of his symptoms it was decided to perform an exploratory laparotomy. On account of his bad colour oxygen was administered, but with no good result.

Chloroform was chosen as the anæsthetic, and was administered with a Vernon-Harcourt inhaler, oxygen being given through the air tube. The cyanosis still increased, so a Schimmelbusch mask with lint was substituted for the inhaler. Induction was very slow, and the corneal reflex was not abolished for twenty minutes, oxygen being given continuously with the chloroform. The respiration became Cheyne-Stokes in type. There was no struggling, and the pulse kept good.

Nothing was found in the abdomen to account for the symptoms, and the wound was being closed when, during one of the apnœic periods of the breathing, the pulse was found to get suddenly weak, and then stop. Artificial respiration and massage of the heart through the abdominal wound were tried without avail.

On post-mortem examination the pericardium was found to be somewhat distended, and to contain nearly half a pint of blood-clot. A small aneurysm, the size of a walnut, was found in the ascending aorta on its anterior aspect, the lowest part being situated about a quarter of an inch above the aortic valve. There was very little laminated clot in the aneurysm. At its lowest part it was very thin, and communicated (by a small opening which just admitted a probe) with the pericardium. The heart was almost empty, there was no valvular disease, but slight hypertrophy of the right ventricle. The large veins in the thorax were distended with blood. There was considerable atheroma of the aorta.

This case is interesting from a surgical point of view, but especially so to the anæsthetist. There can, I think, be little doubt that death was caused by the rupture of the aneurysm into the pericardium, the heart thus being prevented from doing its work by the mechanical obstruction, and this probability is strengthened by the fact that the heart was found quite empty, while the large veins were distended.

The increasing cyanosis in spite of the continuous administration of oxygen would seem to point in the same direction. The almost complete absence of physical signs, which were of help in diagnosing the condition in the chest, is another point of interest.

The sudden evacuation of a large quantity of pus from the pleural cavity, especially the left, may result in death.

For example, a child has been suffering from a left empyema which has gradually increased till the heart's apex has been displaced, perhaps to the right of the middle line. An operation is decided upon, the patient anæsthetised, the pleural cavity opened, and the pus allowed to escape rapidly. In a few seconds the patient dies.

It would seem that death in these cases is due to the sudden relief from the pressure of the fluid on the heart. The muscle of the heart is in poor condition through the septic state of the patient, and it is not improved by the anæsthetic circulating in the blood, though in the majority of instances I do not think that this is the most important factor. There is often some infusion into the pericardium as well, and altogether the heart is put to a great strain to do its work at all. It has got more or less accustomed to its new position, and when this is suddenly altered, or at any rate the pressure on it is suddenly removed, the change is too great and it gives out.

Some surgeons place a sponge or pad over the opening so that the pus may escape slowly; but though it is hardly in my province to criticise any surgical procedure, I believe that the best results will be obtained in these cases by slowly draining off the pus with an aspirator, or better still with Turner's tube; and if a larger opening is found to be necessary, or some portions of rib require excising, that this should not be done for twenty-four or forty-eight hours, when the heart has had time to become more reconciled to its altered conditions.

Cerebral hæmorrhage may cause death during anæsthesia, and this is most likely to occur when ether is given to a patient with advanced atheroma of his cerebral arteries. Dr. Dudley Buxton has avoided this accident on two occasions, for the patient to whom he was to have given ether died from cerebral hæmorrhage a day or two before the operation.

When death occurs during the course of a long and serious operation it may be produced by a combination of causes, such as the effect of the anæsthetic, the loss of blood, or the shock of the surgical proceedings; and in a given case it may be almost impossible to decide which is the most potent factor; but on the other hand, in other instances, one of them can be definitely assigned as the primary cause.

Putting aside the question of the anæsthetic, there is no doubt that hæmorrhage alone is sometimes fatal. When, for instance, during an attempt to remove a cancerous kidney the inferior vena cava has been torn across and death has occurred in less than two minutes the cause is sufficiently obvious, but a more gradual loss of blood may also be fatal. An operation for the removal of malignant glands of the neck proved very difficult and tedious, and hæmorrhage, though never profuse, was almost continuous. When this began to affect the patient's general condition transfusion was begun and continued for a long time, the man eventually dying from gradual hæmorrhage.

The shock of an operation is, of course, diminished by anæsthesia, but even profound anæsthesia does not always obliterate it. It is often noticed when a large tumour of the breast is stripped from the chest-wall, and also in operations on the brain, and the genital organs. It may be so severe that it actually causes death; and a case has recently been related to me by one of our most skilled anæsthetists, in which an elderly gentleman died suddenly during the removal of an enlarged prostate; there had been no severe hæmorrhage, and no overdose of the anæsthetic.

A comparatively new factor in the causation of death must at least be mentioned; I refer to the "status lymphaticus."

This condition is more widely recognised upon the Continent and in America than it is here. But during the course of this year the reports of several inquests have given the status lymphaticus as the cause of death.

This condition is not very definite, but it is said to consist chiefly in a marked hyperplasia of all the lymphoid structures in the body, and to be recognised by an enlargement of the tonsils, the lymphatic glands, the spleen, frequently the thyroid gland, and the thymus gland is always unusually

large. The condition has sometimes been described as the "status thymicus."

There is no doubt that sudden death has often occurred without any obvious and sufficient cause, as, for instance, when a child, apparently perfectly healthy and playing with its toys, turns over in its bed and dies; and there is also no doubt that in many of these perplexing cases the post-mortem examination has revealed this condition of a large thymus gland with enlarged tonsils, large spleen, etc.

But the manner in which death is actually produced is hard to determine. Two theories have been advanced, one that the large thymus gland causes death by mechanical pressure on the trachea, and the other that it is produced through a toxæmia from excessive secretion.

Up to the present time this condition has generally been recognised only on the post-mortem table, which has given cause for critics to compare it to "that blessed word Mesopotamia," when it is advanced as a cause of death under an anæsthetic. But lately an important paper has been read by McCardie before the Society of Anæsthetists, in which the literature of the subject, together with the personal experience of the auther, form such strong arguments that, at least, they merit attention.

In the month of May this year an infant, æt. 9 months, was anæsthetised with chloroform at the London Hospital, while the electric cautery was applied to a rapidly-growing nævus of the back. A similar operation had been performed eight weeks before, and then all had gone well.

The anæsthetist was the same on both occasions, and had given anæsthetics for many similar operations. The child was doing well, respiration was perfectly satisfactory, the colour was quite good, the pulse good, and the anæsthesia light, the corneal reflex being present. The needles had been in about twenty minutes, and in five minutes more were to have been removed, when to the amazement of the anæsthetist and the physician in charge of the department, who was watching the child, it suddenly died. Dr. Morton assures me that an overdose of the anæsthetic could not possibly be the cause of death, as no chloroform had been dropped on the mask for some minutes.

At the post-mortem examination it was found that the thymus was very large, the size of a crown piece, and was lying on the large vessels of the neck. It was six times as large as the thymus of a child of the same age whose body happened to be in the post-mortem room at the same time, and who was considered to be normal. There was enlargement of the tonsils, and the cervical mediastinal, bronchial, axillary and inguinal glands. The faucial glands of the tongue were large and the size of a pea. The Peyer's patches and the lymph-follicles of the ileum were large, red, and swollen. Altogether the pathologist considered that the body presented the typical signs of the "status lymphaticus."

I fear, gentlemen, that I have wearied you enough in this recital of some of the ways in which death may occur during anæsthesia. But, though all the causes may not have been enumerated, I hope that there have been enough for the following conclusions to be fairly drawn:

First, that when death occurs while a patient is under the influence of an anæsthetic, it is not by any means always due to the effect of that anæsthetic.

Second, that when a death does occur under these circumstances, it is most important that a careful post-mortem examination should be made; and perhaps, as a third conclusion, that in some instances the actual share of the anæsthetic in the death may be so extremely difficult to determine, that the help of the average coroner's jury may be almost ludicrous, and we may wish for some method of conducting an inquiry into these cases which offers the prospect of a more useful result than is to be obtained at an inquest, quite apart from the harm that may be done by the sensational reports published in the modern press.

December 21st, 1908.

Transplantation of Vessels and Organs.—Capelle reports results of extensive experimental work in this subject. Implantation of pieces of arteries for artificial defects in other arteries was followed by good functional and anatomical results when the same animal species was used for the operation and for procuring the material. When other species of animals were used the sections of the vessels functionated properly, but after a time showed various stages of degeneration. Sections of veins were found to serve as good substitutes for arterial defects.—*Medical Record*, vol. lxxiv, No. 23.

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NEURASTHENIA IN WOMEN.

A Clinical Lecture delivered at the Polyclinic.

By G. E. HERMAN, M.B., F.R.C.P.,
Consulting Obstetric Physician at the London Hospital.

GENTLEMEN,—As, in a lecture on neurasthenia in women, I must trespass on the domain of neurologists, I gladly admit that there are more competent speakers on the subject than I am. A knowledge of this subject is of importance to anyone who practises gynæcology. I should think I prescribe sodium bromide for three fourths of the patients I see. I saw a paper by an American gynæcologist, after I had for myself found the value of this drug, in which he spoke of it as a sort of specific for the minor diseases of women.

To illustrate my subject I will first call your attention to writings by some distinguished gynæcologists of the past: for disease remains the same, although opinions about it differ.

I have here the works of Dr. Gooch edited by Dr. Ferguson and republished by the New Sydenham Society in 1859. I will read you a part of the prefatory essay, in which the Editor gives a precis of Gooch's writings. Having stated that a very original chapter on the subject of disease of the nervous system was given by Gooch thirty years before under the title of "The Irritable Uterus," Dr. Ferguson says:

"I believe he was the first to call attention to a malady of greater import than he himself suspected, and which, even yet, is regarded as almost an isolated disease, having little connection with those uterine affections, now so rife and so well known. This comparative neglect on the part of the profession is attributable, mainly, to the belief that Gooch had not all the elements of a just diagnosis, which have existed since his time, as afforded by the 'speculum.'

"Gooch describes a malady, of which the

essential symptom is pelvic pain, subsisting even for years, thoroughly incapacitating the patient for the performance of many of the duties of life, and yet unaccompanied by any appreciable structural change. The disease thus described is an undoubted reality: many of those from whom Gooch took his views I have seen, and some few are yet alive to prove how long this obscure malady adhered to all the modifications of structure and function incident to advancing age. I have repeatedly examined with the speculum the seat of pain, and as often been satisfied that it is independent of any vascular complication. In those who are most obnoxious to this form of the disease there is frequently an hereditary taint of gout or rheumatism, or they are the offspring of very nervous parents, themselves exhibiting the nervous temperament in excess. One of the seats of this neuralgic malady is the vagina itself, which is so exquisitely tender as to render intercourse intolerable; indeed, in several instances this condition has led to separation, and the unbalanced nervous power has been the index to greater evils, merging in insanity. It is this form which Gooch has described so ably; separating it from all inflammatory disease, whether chronic or acute, and, by analogy afforded by similar affections in other parts of the body, classifying it with disorder of the nervous function." He goes on to say: "There is, however, another form, or rather another degree of it, which he has not described, but which, in our time, all have had opportunities of witnessing who have noted those instances, which have of late been so largely multiplied, and which are the despair of the practitioner and the bane of many a domestic circle. In this series the purely nervous aspect of the malady is masked by some obvious change in the uterus or its appendages; but this change is by no means a constant one, either in its seat, extent, or nature. Sometimes there is a congested condition of the uterus, altering its shape into that of a retort; the enlarged and curved fundus being exquisitely sensitive of pressure. At other times the cervix, or some portion of the uterine walls, is the seat of congestion, of varying consistency, and of pain. In other instances the uterus may be entirely healthy, but the pain is referred to either ovary, or to some obscure spot of the pelvis itself. I have known the same general train of symptoms co-exist with every form of uterine ulceration, and without any of them; with every degree of uterine infiltration, and without any one of them. In a word the local changes have been the fluctuating, the nervous affection the constant element: in it, therefore, and in no doctrine of a phlogistic origin, can I place the essence of this strange disease. Under a notion of its local origin I have now had abundant opportunities of seeing the effects of local treatment, ranging between the use of the actual cautery, through various modes of caustication to that of a simple astringent, or even watery injection: all have failed to cure, and often even to alleviate the pain. Those who are thus affected go from specialist to specialist, and generally, after years of trial and endless expense, subside into invalid habits, unrelieved; or they gradually wear out the disease."

You see Ferguson did not know anything about vaginismus, which had not at that time been described, or about uterine displacements. But the point is that he recognised a group of nervous symptoms which sometimes occurs with uterine disease and sometimes without any change in the uterus whatever.

I now come to Gooch. He begins by saying: "The disease, which I have ventured to call the irritable uterus, is a painful and tender state of this organ, neither attended by, nor tending to produce, change in its structure. It is now between fifteen and twenty years since I began to notice this disease, and since then I have seen several cases every year. At first it puzzled me much; I had not seen it described in books. I took it for chronic inflammation, which would end in disorganisation, probably of a malignant kind; but experience, whilst it taught me that it was a very intractable disease, taught me also that it was not a disorganising one. I became familiar with its obstinacy, and less apprehensive about its result, for I know cases which have lasted upwards of ten years, in which the structure of the uterus is as unaltered now as it was at the beginning of the disease, as far at least as can be determined by examination during life. Although I often find it still an intractable disease, and wish I had a shorter and surer mode of cure to communicate, yet I think it worth describing, that practitioners may recognise it when they meet with it; that they may know what they are to expect in obstinacy,

and what they need not apprehend in the result; what will do harm, what will do good.

"A patient who is suffering from the irritable uterus complains of pain in the lowest part of the abdomen, along the brim of the pelvis, and often also in the loins. The pain is worse when she is up and taking exercise, and less when she is at rest in the horizontal posture; in this respect it resembles that of prolapsus uteri, but there is this difference, that in the latter, if the patient lies down, she soon becomes quite easy; but in the complaint of which I am speaking, the recumbent posture, although it diminishes, does not remove the pain." He goes on to say: "What is the nature of this disease? It is not acute inflammation, for that would run a far shorter course, and end in certain known consequences. It is not chronic inflammation, for that is a disorganising process, and slowly but surely alters the structure of the organ in which it goes on. Both in chronic inflammation and in the disease which I am describing there is a morbid state of the nerves indicated by pain, and sometimes at least a morbid state of the blood-vessels indicated by their fulness; but the substances effused by chronic inflammation show that in this there is something additional in the actions, and consequently in the state, of the vessels. The disease which I am describing resembles a state which other organs are subject to, and which in them is denominated irritation. Thus surgeons describe what they call an irritable tumour of the breast. It is exquisitely tender, and ungentle examination of the part leaves pain for hours; it is always in pain, but this is greatly increased every month immediately before the menstrual period. Although apprehensions are entertained of cancer it never terminates in disease of structure. It is represented as a very common disease. Mr. Brodie describes a similar state in the joints. It occurs chiefly amongst hysterical females; it is attended by pain." The point is this: patients have pain although Dr. Gooch can find nothing the matter with the uterus.

About his treatment he says: "In all these cases in which the pain is perpetual repose should be perpetual."

Summing up, he says: "Some of my readers will think that the name which I have given to this disease is a verbal refinement, and that I had better have called it chronic inflammation; but I have two objections to this: one is that chronic inflammation, though a slower, is as surely a disorganising process as acute inflammation, but this is not." "The other objection is, that the name would be a mischievous one; many men remember the name of a disease only, and forget the history of it, and the term 'chronic inflammation' would lead to a more indiscriminate and active employment of antiphlogistic remedies than is advisable in the protracted cases. Even the benefit afforded by small local bleedings is not uniform; in some cases the pain is worse after bleeding, in some the pain is most severe a few days before menstruation, when the vessels of the uterus are most distended; in others, on the contrary, the pain is worse after menstruation, when the vessels of the uterus are most drained; in some the uterus feels a little swollen, in others not so: it is probable, therefore, that the cases which I comprehend under the term 'irritable uterus' may require to be subdivided into several classes, in one of which congestion is an essential part, and blood-letting and mercury may be apt remedies; in another congestion may be absent, and blood-letting and mercury may be useless and pernicious, restorative means, especially steel, being the aptest remedies. Lastly, another may consist of those interminable cases which nothing relieves, and which, at the end of ten or fifteen years, are as bad as they were at the beginning; in these there may be some disease of structure in a part of the uterus out of reach of examination by touch."

These two physicians both recognised cases in which there is pelvic pain and nervous disturbance that lasts for years, but no uterine disease. Gooch's arguments apply to-day to people who say that endometritis is common in virgins.

I come now to a greater man than either of these—Dr. Addison—whose name will always be associated with the disease of the supra-renal capsules which he discovered. He was not only a great physician but a morbid anatomist. If he did not find anything the matter with the uterus it was not for want of ability to see it. Addison wrote an essay on 'The Disorders of Females connected with Uterine Irritation.' This essay was published in 1830. He begins by saying: "The object of this lecture is to make you acquainted with the morbid effects produced upon

the general constitution, and upon particular parts of the body, by continued *uterine irritation*. In making the attempt some apology is probably due to my excellent friend and colleague, Dr. Blundell, for thus encroaching upon his particular province."

"I will not occupy your time and attention by any elaborate attempt to define the term *irritation*, a term in common use to signify a disturbance in the endowments or functions of a part independent of either actual inflammation or organic lesion. It is not denied that either the one or the other of these states may occasionally attend or result from such irritation; all that is meant by the term is, as I have said, a disturbance in the endowments or functions of a part without either inflammation or organic lesion being *necessarily* present. In thus characterising uterine irritation, therefore, I do not deny that it may be co-existent with, or even give rise to, inflammation or organic lesion; but, speaking generally, I would say that it is found to exist altogether independently of either of these states.

"The most powerful predisposing cause of uterine irritation is *constitutional irritability*, especially in persons naturally of a delicate frame of body—a state rendering its possessor acutely susceptible of impression generally, and which has not infrequently, and certainly not very inaptly, been distinguished by the term nervous temperament.

"The first complaint usually made by a female suffering from this irritation is of feeling *nervous*, probably with a disposition to be low-spirited; but if, in a well-marked case of the kind, you proceed to feel the pulse, and especially if it be your first visit, the chance is that you will perceive a distinct tremor of the hand, and a remarkable acceleration and sharpness of the pulse, evidently arising from mental agitation—a degree of mental agitation so great, in a naturally susceptible female that if you attempt to soothe or encourage her she will begin to sob, her lips quiver, and she bursts into a flood of tears." "She tells you that, without any assignable cause, she gradually declined in health and spirits; that she has lost her wonted alacrity, has become indolent, and is easily fatigued by comparatively slight exertion; that she is readily flurried; that her heart often beats, flutters or palpitates; that the impressions made upon her mind are altogether disproportionate to the causes producing them; that she is very prone to weep, and occasionally experiences sudden and transitory feelings of alarm and dread, especially during the night, without being able satisfactorily to account for them; in short, that both body and mind are in a morbidly sensitive condition, whilst general distress is strikingly depicted in her pale or dejected countenance. If you proceed in your inquiries to ascertain the state of the uterus, you are perhaps informed that she is regular; but if interrogated more narrowly it will almost uniformly be found that she suffers pain, either before or during the flow, in the back or loins, and that in the intervals she is troubled with leucorrhœal discharge." He goes on to describe the derangement of the digestive organs. "The derangement of the digestive organs, to a greater or less extent, is so commonly associated with the general affection I have described that one cannot but conclude that the general affection is most materially influenced, if not in part produced, by it." He next mentions another symptom, namely, hysterical paroxysms. "The whole assemblage of symptoms, the aberration of mind and bodily contortions, constituting, when taken collectively, what in common language is understood by an hysteric paroxysm. There are, however, divers modifications met with—the patient being sometimes seized with violent and involuntary fits of laughter or crying during the paroxysm, at other times she will lie in a perfectly motionless and insensible state, but with a natural pulse, for hours together; or she shall suffer from a more or less perfect paroxysm without its being preceded by any globus whatever." He goes on to speak of the pain felt affecting the abdominal viscera. "The most frequent are:

"First, a pain seated under the left mamma, or under the margin of the ribs of the same side.

"Secondly, a pain under the margin of the ribs of the right side.

"Thirdly, pain in the course of the descending colon.

"Fourthly, pain in the course of the ascending colon, especially towards the right hypochondrium.

"Fifthly, pain affecting the abdomen generally.

"Sixthly, pain in the region of the stomach.

"And lastly, pain in the region of the kidneys, sometimes extending down the course of the ureters to the bladder."

After describing these different pains he says:

"It must, nevertheless, be carefully remembered that all or most of the symptoms I have enumerated, as emanating more immediately from the uterus itself, may, and often do, result from organic changes either in the uterus or in the parts adjacent."

He adds: "I repeat to you, gentlemen, that the condition of the uterus, to the continuance of which I ascribe the various disorders enumerated in this lecture, has in most instances been altogether overlooked."

There is, you will have noticed, a resemblance between the symptoms described by Gooch, Ferguson and Addison. They all say that while pain and other nervous symptoms occur with uterine disease, they often occur without it. To me they are good descriptions of neurasthenia.

I will read you a passage from another writer, Dr. James Henry Bennett. He says: "Extensive inflammatory and ulcerative disease of the cervix may be present for years without giving rise to pain, or to any well-marked local symptom; the only evidence of its existence, especially to a superficial observer"—one who did not use the speculum—"being functional derangements of the uterus, and sympathetic reactions which we have yet to investigate."

He describes the pain "felt behind the pubis, the anatomical seat of the diseased cervix, but more frequently in one or both of the ovarian regions, in the lower lumbar, and in the upper sacral regions. Singularly enough, in nine cases out of ten, it is the left ovarian region alone, and not the right, or both, that is the seat of pain. This localisation of the pain produced by inflammation and ulceration of the cervix uteri in the left ovarian region may be connected with some peculiarity of the distribution of the uterine nerves, but I have hitherto been unable to discover any anatomical reason for the preference thus shown. The fact, however, is undeniable, and renders the existence of a dull, aching, constant, circumscribed pain in the left ovarian region all but pathognomonic of inflammatory disease of the cervix uteri. The pain in the back is of the same dull, aching character. It is sometimes scarcely perceptible, only amounting to what the patient calls a 'weakness,' except, perhaps, after fatigue. In many instances, however, it is very severe, and may be perfectly agonising, incapacitating the patient for any exertion. She feels, she says, as if the back were broken, and she can neither stand nor sit erect with comfort.

"Uterine neuralgia is often accompanied by general neuralgia, or tic, in the face, head, neck, back, chest, or elsewhere.

"In addition to the lumbo-sacral, ovarian, and hypogastric pains which more peculiarly characterise inflammation and ulceration of the uterine neck, there are often other pains present, which must be attributed to the same cause.

"The constitutional reactions produced by inflammation and ulceration of the neck of the uterus, which form one of the most important features of the disease, have not hitherto been clearly elucidated. These reactions, taking place principally through the sympathetic system of nerves, may be aptly designated the sympathetic symptoms."

Then, after describing the various kinds of indigestion, he goes on to say: "Severe thoracic pains, dyspnœa and other chest-symptoms, however, are sometimes present in females suffering from uterine inflammation, as the result of pulmonary disease." As to general nutrition he remarks: "The patient loses flesh, becomes emaciated, pale, sallow, languid, and weak; falls, in a word, into a more or less marked anæmic state."

He gives the cerebral and spinal symptoms: "Intense headache, great depression and lowness of spirits, and groundless terrors, experienced not only during the night, but even during the day. The cephalalgia may exist in any part of the head, but it is principally observed, as I have stated elsewhere, at the summit and over the forehead. The pain felt at the top of the head is often compared to a heavy weight pressing on it. The mental depression experienced by the patient is often extreme, and not infrequently accompanied by delusions or hallucinations, and by the fear of insanity. This fear is not altogether unfounded, especially where insanity exists hereditarily, the uterine disease, *if unchecked*, in such cases sometimes terminating in the wreck of the mental faculties.

"The special senses are not infrequently affected, and principally the sight and hearing. . . . The cutaneous sensibility is sometimes much exaggerated all over the body, in isolated regions, or

on the left side only. . . . The difficulty in walking and standing which is so frequently observed when the uterus is the seat of inflammation. . . . The various nervous manifestations to which the term hysterical is familiarly applied, are frequently met with in patients suffering from chronic inflammatory disease of the cervix. . . . I have met with several very remarkable cases in which the all but complete loss of the voice, which could only be heard in a whisper, was evidently the sympathetic result of uterine inflammation. . . . But little refreshing sleep is obtained by a person labouring under this disease in a severe form."

I have read quite enough to show you the identity of the symptoms which Bennett observed and which *to him* were those of inflammation of the cervix, with the symptoms described by Gooch and Addison.

I turn to another later writer, Dr. Graily Hewitt, who wrote a book called 'The Mechanical System of Uterine Pathology.' In his view these same symptoms were all due to displacement. He says: "The first remark to be made in connection with this subject is, that cases vary very much in severity, and too much has been expected in regard to uniformity of symptoms when the conditions were not uniform." Referring to reflex phenomena in connection with disorders of the uterus he states: "These reflex phenomena are very important, and the highest interest attaches to any discussion concerning them. In the limited space at my disposal I fear that scant justice can be done to this part of the subject. These reflex phenomena I propose to consider under five heads: (1) Sickness or nausea; (2) hysteria; (3) convulsions; (4) cephalalgia; (5) melancholia." I need not read any more than that.

You see that all these gentlemen agree in describing the seat of symptoms which they met with as in their opinion due to slight morbid changes in the uterus and very often met with where there was no disease at all. Bennett put it down to erosion of the cervix; Gooch and Addison say they have found the same symptoms when the cervix is perfectly healthy. There is a phrase often used in connection with various morbid conditions—"protean symptoms"—which means that with the same local changes the symptoms vary to an extreme degree. The fact is that the patients are patients with neurasthenia, and the uterine condition is a small part of the case. Neurasthenia is what old writers called "irritable weakness." The strength of the nervous system is reduced, and the patient cannot resist pain as a healthy person can. Probably some of you have suffered from influenza, and you will remember that when you go to bed with influenza contact with the sheets is extremely disagreeable because resistance to pain is broken down by that disease. In these neurasthenic patients the nervous system is weak, and therefore the reflex irritability is increased. They are not hypochondriacs; they do not fancy they have a disease when they have not one. Neurasthenic patients have quite enough of disease without fancying it.

As to the causes of neurasthenia, in some cases, no doubt, as one of these writers remarks, the weak nervous system is inherited. Those who have to deal with nervous weakness in children find that if they can get the patients young enough they can do a great deal by education for the most hopeless children. I think that inheritance, although it undoubtedly does play a part in the causation of neurasthenia, yet plays a small part. You must remember, with regard to a child born of very nervous parents, that not only does it inherit some of the tendencies of its parents, but may be subject to a most injudicious education.

I have here a report, which appeared in one of the daily papers—the 'Standard'—of a lecture entitled "The Curse of Education," given on Saturday evening at a meeting at Cambridge by Mr. Harold E. Gorst. First of all I should say that what Mr. Gorst speaks of as "education," I should call it *information*. Education consists in training the faculties to observe, to think, and to do. But information, miscalled "education," consists in making the pupil learn by heart other people's opinions. The report stated: "Mr. Gorst protested against the monotonous process of cramming in our system of education. He gave several grounds for stating that our education system was a curse, among them being that it helped to fill the country with misplaced individuals, and in that way ensured a supply of mediocrity to every profession and colony. The University graduate, as such, had no practical accomplishments. It was a danger to the State to recruit its public servants by means of competitive examinations. The artificial process

of cramming in some cases caused actual injury to the brain." Cramming means learning by heart other people's opinions instead of learning to observe, and think, and do. "I do not deny," said Mr. Gorst, "the possibility of a man being crammed successfully for an honours degree and afterwards throwing off his incubus of knowledge even to the extent of becoming a Cabinet Minister." These remarks only apply to a small class. This is the more important part of his lecture: "Our educational system was a cruel deception of the poor, who thought their children were being fitted for something, and would be enabled to rise in life, whereas the children of the working classes were fitted for nothing, and only learnt to despise their station in life. Our system of education helped to make criminals. . . . Real education would encourage the utilisation of the brain for purposes of thought and reflection, instead of training to make it a repository for storing van-loads of useless knowledge." I agree with him. To make ill-fed children learn words by heart is a waste of time, and a mode of producing neurasthenia. Mr. Gorst would have the children taught to do something, and to learn to do anything well they must learn to observe. There are savage tribes in whom the faculty of observation is more highly trained than that of Europeans—for instance, the black trackers in Australia.

There are certain sexual causes of neurasthenia. The practice of preventing pregnancy often develops neurasthenia. If the sexual appetite is frequently excited and never satisfied the woman will lie awake and lose sleep and become neurasthenic. Among the working-classes a great cause is the anxiety and worry connected with the bearing and rearing of children. A mother who can give the baby to a nurse and have leisure and undisturbed sleep is in a different position from that of a poor woman who has to nurse the baby, to work in her home, and is kept awake at night by the squalling of the child. In this case the nervous strength of the mother becomes reduced by the excessive calls made upon her. A woman unhappily married often becomes neurasthenic.

The shock and strain involved in operations is a cause of neurasthenia. The late Mr. Knowsley Thornton used to tell his patients that they would not be themselves for twelve months after ovariotomy. I think he rather over-stated it, but it was honest to his patients and safer to err on the right side.

I have but little time to speak of the symptoms. The patients will tell you they are low-spirited and easily made to cry. They will tell you that they start whenever the door is slammed, and that they cannot do much either physically or mentally. They are easily frightened. Because they have so little energy they get to be wrapped up in their own affairs and have little sympathy and consideration for other people. They almost always sleep badly. Want of sleep acts disastrously upon the nervous system. I have read somewhere that dogs can be killed more quickly by keeping them without sleep than by starving them. Neurasthenic patients have morbid sensations either in the head, eyes, or limbs. There is a kind of headache produced by want of sleep. The patient complains of an unpleasant sensation on the top of the head—sometimes of a weight, sometimes of a running or trickling on the top of the head. There is nearly always dyspepsia and discomfort or nausea after food. In certain cases mobile viscera are found. I do not think that this mobility of viscera has anything to do with neurasthenia. If the patient had remained well, in a great many cases no one would have ever found out that a viscus was movable.

The patients are subject to hysterical fits. Although certain seizures are called "hysterical" they have nothing to do with the condition which neurologists call hysteria. We know more about hysteria now than was known in the time of Addison, Gooch, or Hewitt. It is a disease having definite physical signs. In many cases of hysteria a hysterical fit never occurs, whereas hysterical seizures with laughing and crying and the passing of limpid urine at the end are common in neurasthenia, but only occur in the minority of cases of hysteria.

There are pains which the writers I have quoted mention. The pain of neurasthenia is commoner on the left than on the right side. That is because the left side of the body is weaker than the right in resisting the pain as well as in muscular power. It is found in cancer that pain is commoner on the left side than on the right.

All these symptoms occur without uterine disease as well as with uterine disease. Diseases which, as these writers found, cause "protean"

symptoms, are only slight diseases. If the patient had been strong she would not have gone to the doctor at all; she went because she is suffering from neurasthenia.

If there is any local disease, for instance, chronic inflammation of the ovary or tubes, displacement, or any condition which is causing pain, the presence of persistent pain in time wears down the resistance of the nervous system and makes the patient neurasthenic. The condition which causes the pain should be cured.

These patients sleep badly. The best way to make them sleep soundly is to give 15 grains of sodium bromide three times a day. After a fortnight a young neurasthenic woman will tell you she is sleeping well and feels better.

The great thing to do in the treatment of neurasthenia is to give the patient rest, food, and sleep, and to remove the local pain. If you do these four things you will cure the patient. In bad cases you must have resort to hypnotics. There are many of them. Some, if you give them for a long time, do harm. Others may do harm—I do not know. But, so far as I know, trional in doses of 15 grains can be taken many times without harm. I know no harm from veronal in doses of $7\frac{1}{2}$ grains. Nor have I heard of harm resulting from the frequent use of chloralamide. Harm will result from excessive doses of any drug.

Nearly all patients with slight uterine disease and neurasthenia, if taken into a hospital, allowed to sleep soundly and given plenty of food, will get well.

A great deal has been done within the last quarter of a century in rest cures, isolation, massage and electricity. You have to recollect that massage and electricity means skilful attendance, with expensive apparatus. Isolation also means expense. So that, as a Scotch doctor cleverly put it to me, "the patient gains a stone and loses two hundred pounds." Dr. J. M. Baldy, speaking at the Obstetrical Society of Philadelphia, told his audience that "beyond falling into the hands of an operating gynæcologist, he knew of but one equal misfortune for these much-afflicted individuals, and that was to fall into the hands of a rest-cure neurologist." When you think that there is no hospital in London that would accommodate all the poor women who are suffering from neurasthenia, it is clear that isolation, massage and electricity are out of the question with regard to them. The use of massage is to promote the circulation and make the patient more comfortable. The use of galvanism is to prevent temporary weakness of the muscles, which always occurs when a patient has been in bed for some time. After an ordinary illness, when a man gets up who has lain in bed two or three weeks, he always feels weak about the legs. Massage and galvanism are used to prevent that feeling of weakness. But these forms of treatment are not in my judgment so important as to make it worth while to call upon the patient to put himself at some considerable pecuniary sacrifice. Isolation is not so important or beneficial as some people think. I have known patients who resented isolation most bitterly; solitude to them was extremely unpleasant.

There is one form of neurasthenia in which I doubt whether the name is quite appropriate—cases in which the patient suffers from excessive irritability; is, to use a colloquialism, always on the go, cannot keep herself still, and yet does not appear to be weak, though she complains of this or that symptom. With regard to these excessively irritable patients I have certainly found arsenic do them good. Dr. Gooch had to treat neurasthenic patients without bromide because it was not then known. There are cases in which the patients are plump and well nourished and sleep soundly, but nevertheless complain of some of the various symptoms which I have mentioned. In such cases the treatment is extremely difficult.

December 28th, 1908.

Action of Roentgenised Serum in Leucocytosis and Leukæmia.—Clinical observations on man show that injection of serum from a healthy man who has been submitted to the rays causes marked leucolysis. With intravenous injections this is lost after twenty-four hours, but with hypodermic injections the condition is produced more slowly, but is more efficacious, lasts longer, and is progressive. This action is accompanied by a general improvement, increased elimination of uric acid by the kidneys, and reduction in the size of the spleen. Probably this serum contains specific antibodies produced by the stimulus of the X rays upon the tissues, which cause all these good effects on the leukæmic condition.—*Medical Record*, vol. lxxiv, No. 24.

WITH MR. BLAND-SUTTON AT MIDDLESEX HOSPITAL.

III.

GENTLEMEN,—To-day I propose to discuss with you the way in which cancer patients die, especially patients with cancer of the breast. Cancer of the breast, as I told you before, has been known for centuries; the part has always been so open to observation; it is, indeed, the organ from which we derived primarily all our knowledge of the course, structure, treatment and terminations of cancer. The interesting thing with regard to cancer of the breast is this: it is not a vital organ; it is an organ which the patient can easily do without, and therefore when it is the seat of cancer you can watch the course of the disease in its purest form. You will remember that when we were talking about the general characters of cancer, its microscopical features and its mode of spread, I was anxious to point out to you that the cancer itself, the primary focus, cannot be regarded as a rapidly fatal disease; cancer itself is dangerous and fatal on account of the peculiar way in which it disseminates itself, and also from the method in which it lays the patient open to septic infection. I have already told you that the majority of patients who die from cancer die from some secondary consequence of the disease rather than from the primary disease. The effects of the primary disease can be studied better from cancer of the breast than in any other organ, and some people are averse to having anything to do with doctors, even years before Christian Science was invented, and who look upon doctors as malicious people, and keep carefully away from them. It is in these cases that you can study the natural history, so to speak, of cancer. Some of these cases are very interesting to study because I shall try to show you that the primary growth sometimes undergoes spontaneous cure. This is a matter of very great interest, because there are many people nowadays who look upon cancer as a parasitic disease; and it may be compared in many ways with syphilis, which, as you know, starts from a primary sore or ulcer, and in the course of a certain number of weeks, whether the sore is treated or not, that primary sore heals. But it is replaced by a well-marked scar, which persists throughout the whole of the patient's life. Although the primary sore heals the system becomes infected, and later on a number of secondary lumps develop in the tissues and organs of the body, which are known as gummata. In many ways cancer approaches nearer to syphilis as an infective disease than any other. The great point of difference between them is this: that in the case of syphilis the disease in remote organs, the gummatous lumps, are not produced by the transference of any tissue particles from the seat of the disease, but it is the active infective agent, probably the spirochæta, which produces it. And therefore all the tumours, wherever the lesion of syphilis occurs, whether it be on the tip of the nose or the tip of the penis, or on the finger or on the buttock, the secondary lesions in the patient's body will be precisely the same, whereas in the case of carcinoma the secondary lumps which form in the body agree in character with the structure of the tissues in which the primary growth arose. That is the important distinction. What I want to impress upon you is this: that it is rare that the primary focus of cancer, particularly as far as the breast is concerned, is the cause of death; it is the secondary results which arise from that primary focus which produce such disastrous results to the individual. In order to show you how long some of these cases of cancer may exist in individuals, I may mention that there is more than one case in the records where patients have lived in this institution for twenty years with slowly progressive cancer of the breast.

Such cases are known as atrophic or withering cancer of the breast, and although the disease slowly destroys the nipple and the underlying glandular tissue, and progresses for ten, fifteen, or twenty years, the ulcer does not get any larger. The breast is continually shrinking away, and even both breasts may be destroyed. Then some intercurrent malady, like pneumonia, or some other lung trouble, will carry the patient off. There is nothing so striking as to see a patient in the wards and notice the slow way in which this form of cancer grows and causes the patients very little inconvenience, no pain, and they are able to enjoy life much as other people do. Every now and then a patient will come to you, when she has

heard of the death of some friend from cancer, and in the most confidential way she will tell you that she has had an ulcer of this kind for five or six or eight years, and that she thinks it is time it was attended to. You do nothing for these cases; operation is not required for them, and those patients will live a very long time. But the ulcer never disappears; it is always slowly progressing and slowly shrinking the breast up, and it is as surely destroying also the tissues around. Unfortunately, however, all cases of carcinoma do not run this slow course. The younger the patient the more rapidly the disease grows. As far as I know, no cancer of the breast has ever been recorded in a patient younger than 15 years of age. There are very few cases of cancer of the breast recorded before the age of 25. The bulk of breast cancers are met with in women between 35 and 50 years of age: it is in those fifteen years that the larger proportion of breast cases come under observation. But as a matter of fact cancer of the breast may be met with at any age, even in women 90 years of age. The younger the patient the more rapidly the cancer grows, and the more rapidly it disseminates. The worst condition in which you meet with cancer of the breast is in women who are suckling. It does sometimes happen that a patient with cancer of the breast becomes pregnant; then the cancer will grow with tremendous rapidity; such cases are referred to in text-books of surgery as acute scirrhus, and the skin of the breast is quickly involved, the lymph-glands are infected, and the patient dies very rapidly from the dissemination of the disease. Cancers are badly supplied with blood; they are very rich in lymphatics, and the breast, like other cutaneous organs, is very abundantly supplied with lymphatics, and these lymphatics communicate with the lymph-glands in the axilla and in the mediastinum. There is also a rich plexus of lymphatics permeating the whole of the fascia which covers the body, and by means of these lymphatics the breast is in communication with almost every part of the body. The first thing that happens when a patient has cancer of the breast is that the cancer-cells make their way into the lymphatics, and the lymphatics which will be first affected will depend on the position in the breast of the primary focus of the disease. When the cancer affects the breast at the base of the nipple, the lymphatics in the axilla are soon infected; when the cancerous focus is on the sternal side of the nipple, and particularly in what is spoken of as the sternal process of the breast, then the cancerous material will find its way into the lymph-glands which lie in the mediastinum. They will pass along the lymphatics which pierce the intercostals near the border of the sternum. I want you to understand, too, how these cancerous cells involve the lymphatics. It is not merely that the cancer-cells find their way into the lumen of the lymphatics and then float along like a cork on a stream, or like fish in a pond; that is not the way they go. One of the most striking features of a cancer-cell is its power of invading and eroding any tissues with which it comes in contact. So great is this power of the cancer-cell to erode living tissue that it will eat up and form large holes in bones, as these specimens before you testify. Cancer-cells rarely invade the walls of arteries, but they quickly implicate the thin walls of veins and lymphatics, and in well-marked conditions the tissues of these vessels will be replaced, and their lumina obliterated by a solid cord of cancer. We must now consider the effects on the lymph-glands. In a normal condition the lymphatic glands are little specks not larger than a pin-head. If you were making an ordinary examination of the axilla in the operating theatre you would find the lymphatic glands in the normal way are quite small things, very little larger than a pin-head. But when they become infected with cancer they may attain to even the size of chestnuts, and then they will form a large bunch in the axilla, which, in the later stages, will be as big as a fist. When the lymph-glands enlarge they are in the early stages easily separable from each other, but in the later stages they may become so stuffed with cancerous material that the capsules burst and the glands form a conglomerate mass. The limits of the separate glands are usually destroyed, and then the cancer implicates the skin of the axilla and ends in ulceration, and the ulcerating lump in the breast and ulcerating mass in the axilla tend to fuse together and form a horrible chasm, and on its floor in advanced cases you will see the axillary vessels pulsating. In some cases the axillary vein is broached by the ulceration, and the patient will suddenly get free hæmorrhage and bleed to death. It is an easy ending, for when these patients are slowly bleeding to death they will say

they never felt so comfortable during the whole course of their disease. I have seen several instances of this, that I am sure it is no use trying to prolong their lives by permitting an energetic house-surgeon to apply a ligature to the bleeding vessel.

But there are other ways in which cancer cases die. After the lymph-glands in the axilla are plugged up, then the infection extends above the clavicle and infects the supra-clavicular glands, and this leads to infection of the thoracic or the right lymphatic duct, according to the side affected, and the cancer material finds its way easily into the blood and is distributed to the lungs. When the lymphatics lying in the mediastinum become affected, as they often do, then you get respiratory difficulties, because the enlarged glands in the thorax press upon the trachea, and a fair proportion of patients with cancer of the breast die from suffocation owing to the pressure of enlarged glands on the trachea.

These are the chief ways in which the lymphatic glands may cause death in cases of cancer. While these big glands are enlarging during all this time, the permeation is going on into the minute lymphatics lying in the fascia, which covers the thorax and underlies the breast and covers the abdomen. And I daresay you will be surprised sometimes in the post-mortem room, when you come and see bodies examined of patients who die from cancer, to find large masses of cancer in the liver and large masses of cancer in the ovaries and omentum. And it was thought for years that this infection of the liver and of the ovaries was due to transportation of cancer-cells from the primary focus by means of the blood-stream into these distant abdominal organs. But Mr. Handley, who has investigated this matter, shows that it is not so. He tells us that this infection of the abdomen occurs in a very insidious way. The cancer material finds its way down the lymphatics underlying the fascia of the body and makes its way towards the epigastrium. And if you think of the relation of the fibrous tissue at the apex of the sternum you will see what a number of fibrous structures come into relation at that point. There is the fascia, which overlies the body generally, immediately beneath the fat. Then there is the linea alba, where all these abdominal muscles meet together, with the sheath of the rectus and all the aponeuroses connected with the abdominal muscles. And there at the point of the xiphi-sternum, where there is nothing but a big collection of fascia. Mr. Handley has shown that in these chronic cases the cancer-cells permeate the lymphatics, they travel down the fascia, they meet at the epigastrium and pass along down the round ligament of the umbilicus, and the whole of these soft tissues become infiltrated with cancer-cells. They then infect the lymphatic plexus lying in relation with the peritoneum, and these cancer-cells fall into the general cavity of the abdomen; they drop on the liver and omentum and there take root and grow into secondary masses. The peritoneum always contains a certain amount of fluid or lymph, and these cancer-cells get into the peritoneal fluid and are conveyed by it into the recesses of the pelvis and lodge on the ovaries and the vascular uterus, and there they grow into big cancerous masses. It seems as if the ovary is particularly favourable for the growth of cancer-cells. Here is a large ovary; it was almost as big as a football, and is converted into a large multinodular tumour. All these little lumps are secondary nodules of cancer. And here is the mass in section, showing how it is made up of a multitude of glandular nodules of cancer. I used to be puzzled by this. One can understand it being disseminated by the blood-stream and getting into one ovary, but in so many instances both ovaries were affected, even when one could not find secondary nodules in any other part of the abdomen. We could not understand why the secondary nodules appeared in this way in the ovary. That is the explanation: the peritoneal fluid seems to keep alive the cancer-cells; they drop in through the fascia of the peritoneum, the cells are shed into the peritoneum, and they lodge on the liver and intestine and omentum, and the ovary seems to be a particularly favourite seat for them to flourish on. And those of you who are dressers, I ask you, when you see advanced cases of cancer, to examine the umbilicus; it is well worth while. You will often see that when there is what is called epigastric invasion of the abdomen by secondary cancer, the umbilicus will be retracted and drawn down by the cancer growing along the course of the round ligament of the liver, and it produces an appearance resembling retraction of the nipple. A very large number of patients with

cancer of the breast die from this epigastric invasion; perhaps the proportion who do so is from 15 to 20 per cent. Indeed, Mr. Handley goes so far as to say that this epigastric invasion is so common, and is often seen so soon after the operation, that it should be our duty, when we examine a patient to see if she is fit to be submitted to excision of the breast for the cure of her cancer, to make a pelvic examination, so as to be sure that the pelvic organs are not infected, because if they are it is no use removing the breast.

Another and very dreadful way in which patients suffer is when the skin covering the chest is invaded by cancer. I think this condition of things is more common after operation than it is in patients who have not been submitted to operation. Here is the picture of a patient who had her breast removed. She had a recurrence in the lymphatics of the skin, and in consequence the whole of this skin becomes exactly like leather; it is dotted all over with cancerous nodules in the lymphatics, and they coalesce to form a mass like a leather waistcoat. And the French name for it is cancer *en cuirasse*. But we in England prefer to call it the pig-skin condition. Whenever there is this peculiar condition of skin and the lymphatics of the axilla are blocked up, then the arm begins to swell, and it is always an uncomfortable sign when a patient comes back with recurrence and the arm begins to swell. This goes on until the arm gets to look more like an elephant's leg than a woman's arm; it is so swollen and dense and hard that it can scarcely be impressed by the finger. These swollen arms in patients with recurrent cancer are due to blocking of the right lymphatic duct. It is more common on the right side than on the left, and so heavy are these arms that they produce a lateral curvature of

FIG. 1.—Cancer *en cuirasse* with lymphatic œdema of the arm. From a photograph.

the spine if the patient persists in walking about. At last the arm gets so heavy that the patients are confined to bed (Fig. 1). Some years ago it was the practice to take such arms off. I have taken several off myself for this condition, and in two instances I have even removed the scapula, and the patients have been very comfortable in consequence. I have known a case live two years after such an operation. Still, the condition of these patients is very miserable. Mr. Handley, acting on his idea concerning the spread of cancer by means of the lymphatics, has introduced and practised with success an operation by means of which he makes a new lymphatic channel. He introduces a silk thread into the subcutaneous tissue of the arm, and upwards to the wall of the chest. This thread acts like the wick of a candle, and drains the lymph back into the circulation; it is much simpler than practising removal of the arm. I have seen several of the cases in which this procedure has been carried out, and the patients have been very comfortable. It is a good example of scientific work being put into practice.

Another way in which women die is when secondary cancer occurs in bone. We have always thought that cancer found its way into bone by the blood-vessels. We know now that it is mainly due to permeation. When a bone becomes permeated with cancer any slight effort will cause the bone to break, and cases have happened in the cancer ward where a patient raised her hand to scratch the back of her head, even this slight effort has caused the clavicle to break; or she may be arranging the pillows in her bed and break the humerus, or in trying to get out of bed, or scratch one foot with the other, she will break her thigh bone. This is called spontaneous fracture, and although these bones are permeated with cancer, yet if they are kept in apposition by splint and bandage in the same way as in fractures from other causes union often occurs. Deposits of cancer in long bones after cancer of the breast rarely attain great size, and although entailing strict confinement to bed they rarely produce pain. But there is a condition in which cancer in bones produces agonising pain, and that is when there is a deposition of cancer in the vertebræ. The pressure of a cancerous mass on the roots of the nerves, and on the nerve-trunks as they issue from the intervertebral foramina, produces most agonising pain. This is a practical point to remember, because sometimes old ladies who have cancer hidden away in their breast, which they do not like anyone to see, will consult you because they are unable to pass their water properly, or because they have pain in the legs. Whenever you are called to such a case and you do not find anything to account for the symptoms, always carefully examine the breast. In a certain number of such cases you will find a cancer of the breast which they have said nothing about and which they have had for many years, yet of course they cannot think of any connection between the ulcer in the breast and the pain in the back. When these women become paraplegic in that way there is no operation which can be done for them; they get troubled with bladder symptoms, the sphincters are paralysed and they die from uræmia due to septic pyelitis.

Although we try to treat cases of cancer by operation, the rule in cancer of the breast is to always remove the breast as soon as you recognise it to be cancerous, in the belief that cancer is a local disease. Later on, when you have time to read the history of these things, you will be interested in the accounts of the cancer discussions of thirty years ago, when the greatest surgeons, physicians and pathologists of the day used to argue whether cancer was a constitutional or a local disease. To-day the idea that it is a local disease has all its own way: practically everybody believes cancer is a local disease, and the way to cure it is to remove it as soon as you see it; so long as there is no obvious secondary deposit you may do the patient good. But do not run away with the idea that all the cases you operate upon are going to be cured. Some of those cases are made very much worse by operation, and I tell you why. We do not know the limitations of cancer; it lacks a capsule, and when you take a cancerous breast and divide it with a knife you cannot define its boundaries. This is why a surgeon takes away the whole breast even when the cancer is no bigger than a cherry. If the disease has been in existence some time, and the lymphatics are permeated with cancer, when you come to take off the breast and begin to clear out the axilla and open up fresh tissue, if you are not careful about keeping very wide of the cancer, and if you crush the axillary glands and squeeze the

cancerous material out of them and accidentally spread it about the wound area, you practically sow this freshly prepared ground with cancer-cells, and these cancer-cells will grow, and sometimes the patients come back a few months later with the whole operation area infiltrated and hard with cancer. As a matter of fact in such cases the surgeon has produced a fresh condition of cancer, known as post-operative cancer infection, and there is no doubt that in some instances, where operations have been performed clumsily, and even sometimes when they have been performed with great skill, but particularly when they have been done roughly, the disease becomes much more extensive than it ever was before. We do our best. Some of our patients do remarkably well, some are no better off after operation than if they had been left to run their own course. However thoroughly or early in the course of the disease you remove the breast no prognosis can be made as to the future of the patient. The fact which justifies surgeons in dealing with these cases is that no amount of microscopical examination of the tissues will enable them to make a forecast. In many cases which look clinically unpromising the after-events have proved good, and on the other hand many cases which seemed to promise favourably have turned out badly.

There is yet another way in which these cases die. Cancer implicates the chest wall, or if it recurs it invades the chest wall, and permeates the intercostal muscles and infects the pleura and interferes with the lungs, and then the patient gets bronchitis and septic pneumonia, and pleurisy, and other chest complications which terminate fatally. The one lesson I want you to take away with you is this: it is not so much the primary focus of the disease which destroys life as its power of dissemination, and the remarkable vitality and power possessed by cancerous cells of growing in any tissue of the body, whether skin or bone, brain or liver, retina or ovary.

December 28th, 1908.

We have received from Rebman Limited a copy of Dr. Iwan Bloch's book on 'The Sexual Life of Our Time in its Relations to Modern Civilisation,' translated from the sixth German edition by Dr. M. Eden Paul.

TUBERCLE AND UNDERWEAR.

By LEONARD WILLIAMS, M.D., M.R.C.P.,
Physician to the French Hospital; Assistant Physician to the Metropolitan Hospital.

The strength of tuberculosis resides largely in its power of successful masquerade. It masquerades in general, and it masquerades in particular. It masqueraded for centuries as the child of cold and damp, and succeeded to such good purpose that it established itself as one of the greatest destructive powers on this planet. It succeeds to-day in gaining invaluable time by successfully masquerading, even before the eyes of keen and trained observers, as some transient and harmless malady. It deceives the physician, it deludes the surgeon; above all, it hoodwinks the hygienist.

The hygienist is one who is versed in the science of the preservation of health. Him the demon of tuberculosis has persuaded that the most suitable underwear for people in general, and for the tuberculous in particular, is the material, namely, flannel or wool, which of all others is the least conducive to health, and the most favourable to the development and progress of tubercle. And yet the hygienist is not easily deceived. In the last few decades, by his methods of scientific experiment and patient observation, he has read the secret of several dread diseases. He has beaten smallpox and typhus, he has exposed enteric; and malarial fever he has triumphantly tracked to its lair among the swamps. Nevertheless, tubercle remains his master; to the extent even of causing him to advocate, obstinately and even aggressively, a custom, which on grounds both theoretical and practical, is grossly, ridiculously, and demonstrably wrong. Let us see.

The two main functions of the skin are generally considered to be the preservation of the heat equilibrium and the excretion of certain effete matters in the form of fluid or watery vapour. Assuming that this view of the matter is correct, and assuming also that clothing of some kind is essential to dwellers in the temperate zone, it follows that, of such clothing, the portion to be worn next the skin should be endowed with the two properties which will best assist in the performance of the two principal cutaneous functions. The material should, in fact, be warm and absorbent.

In the temperate zone people suffer from extremes of cold, rarely from extremes of heat. Underwear should therefore be warm. No material is "warm" *per se*. The warmth is necessarily derived from the body, so that a warm material is merely one which does not allow the heat to escape rapidly, a material which is, in short, a bad conductor. Atmospheric air being one of the worst conductors of heat, from this point of view, a very suitable material is one which is so constructed as to imprison a considerable portion of atmospheric air in its meshes.

The excrementitious fluids and vapours which are constantly being given off from the skin should, like all other excrementitious matter, be removed as soon as possible from the cutaneous surface. To this end, it is essential that underwear should be absorbent, and absorbent in a very high degree. If it is not, not only does the excrementitious fluid remain in contact with the skin to irritate it, but the layer of atmospheric air which lies between the garment and the skin itself, rapidly becomes surcharged with moisture and excreted gases. However tightly fitting the garment may be, there is always this layer of atmospheric air between it and the skin, and the skin, be it remembered, is a respiratory organ.

So much is generally conceded; the theoretical propositions are, indeed, beyond dispute. It is only when we come to their practical application that the machinations of the demon of tubercle become apparent. This demon has decreed that flannel or wool (they are the same thing) shall be considered to be the proper material for underwear, and, further, that he who thinks otherwise shall be deemed unorthodox—either a fool or a faddist. Of the two properties essential to wholesome underwear—warmth and the power of absorption—flannel possesses one only, namely, warmth. Of the other, superficially the less attractive, but hygienically infinitely the more important, namely, a high degree of absorptive power, it possesses little or none at all. The demon, however, has contrived to sit at the elbow of all the writers of all the text-books on the subject, and has mesmerised them into applying the adjective "hygroscopic" to this material. Had they not been mesmerised, it is certain that these authorities, some of them at any rate, would have tested the truth of this claim. A very simple experiment would have shown them its falsity and proclaimed the danger to which its acceptance exposes the credulous.

If a piece of flannel is placed on water, it will float for hours before it sinks; indeed, if the flannel be "new," unless the water be agitated, it will not sink at all. Compare this with the behaviour in the same circumstances of a piece of linen, or cotton, or silk. The kerchief made of any of these materials sinks immediately, showing that its absorptive power is infinitely greater than that of flannel. And yet the latter is authoritatively described as "hygroscopic." If the believers in its possession of this property were to substitute a flannel for a linen pocket-handkerchief the next time they suffer from a cold in the head, the experience would probably suggest some more homely adjective. Except in comparison with mackintosh-sheeting and zinc-roofing, flannel is not hygroscopic; in comparison with linen, or cotton, or silk, it is conspicuously and essentially devoid of absorptive power. This refers to new flannel. The washing process to which undergarments are subjected causes flannel to shrink. This means that the air-spaces to which the material owes its "warmth" are gradually abolished by the attentions of the washerwoman, so that a piece of old flannel is neither "warm" in the above sense nor absorbent. From the standpoint of hygienic underwear it has now become not only partly, but wholly and actively, deleterious; a garment made of newspaper could hardly be more so.

In the light of these considerations, the effects of wearing flannel or wool in contact with the skin are not very difficult to estimate. The excrementitious moisture which is constantly being given off from the cutaneous surface cannot find that exit which Nature intended it to find, and it is consequently imprisoned in the layer of atmospheric air which lies between the garment and the skin. This layer, accordingly, rapidly becomes impure and moist. Setting aside the respiratory function of the skin, whose importance, though admitted in theory, is invariably hastily brushed aside as soon as any question of underwear is concerned, it is pertinent to inquire what effect this layer of moist and impure air is likely to exercise upon the two cutaneous functions whose importance is commonly regarded as paramount.

And first as to the heat-regulating function. Whatever the variations in the temperature of the surrounding medium, the body is normally main-

tained at the uniform temperature of 98·4° F. by means of corresponding variations in the contraction and dilatation of the vessels in the integument. To a cold influence they contract, so as to prevent undue loss of heat; to a warm influence they relax, so as to favour evaporation and radiation. The layer of warm moist air which flannel imprisons against the skin seriously interferes with this process, for the cutaneous vessels are in a continual state of undue relaxation; they are being subjected to the same influence as a poultice exerts, and do not therefore contract adequately to a cold influence. That is the reason why flannel-wearers always complain so bitterly of the cold, their pathetically futile remedy for which is to multiply the number of garments of the very material which is the cause of their miseries.

From the point of view of tubercle, both actual and potential, this has a very serious bearing. The tuberculous poison is a powerful vaso-dilator, so that the practice of wearing flannel imitates closely the results which the bacillus itself seeks to effect, results which are therefore presumably the most advantageous to its development. But vaso-dilation means low blood-pressure, and low blood-pressure spells physical lassitude and mental lethargy. In the case of the young, this inevitably leads to defective development, and both in young and mature it provides a fitting nidus for any pathogenic germ which may be watching its opportunity.

The poultice-like effect of wearing flannel induces an enormous excess of cutaneous secretion, and here again we find a very close imitation of the behaviour of the tuberculous poison. In France they recognise that in the case of the potentially tuberculous the great thing to avoid is demineralisation of the tissues, a process which is immensely favoured by the undue activity of the sweat-glands, for which the wearing of flannel is responsible.

The disinclination, however, of the mesmerised hygienist to discuss the question of the skin as a respiratory organ should not prevent those who have escaped the mesmeric influence from looking the matter in the face. Everyone now admits the paramount importance of supplying fresh air in superabundance to the lungs, but where it is a question of the other, and the only other, respiratory organ, the matter of fresh air is considered of no account. The skin is, in point of fact, still treated as the lungs used to be treated in the pre-sanatorium days, the days of the apotheosis of "warmth" and the neglect of purity. Where the skin is concerned the cry is still "warmth" at all hazards; purity is regarded as a fad and physiology as an academic study whose teachings can be considered only when they happen to be in consonance with the requirements of the thermal monster which a stiff-necked and ignorant generation has elected to worship. And this "warmth" which they affect to cultivate is precisely what the flannel-wearers never attain. Their relaxed cutaneous vessels do not, because they cannot, respond to a cold influence, undue loss of heat is constantly occurring, and instead of treating their skins on rational physiological principles, instead of training them to respond promptly and adequately to external variations, they increase the dosage of the vessel-relaxing and health-destroying flannel. Is it any wonder that the children among them have adenoids and are rickety, or that the adults regard a "draught" as their deadliest enemy?

At the root of the enormous amount of educative work which still remains to be accomplished in the fight against tuberculosis lies the question of the hygiene of the skin. The flannel-wearer hates fresh air because it is cold, and so long as he remains a flannel-wearer he is powerless to react to cold influences. He therefore fights stubbornly and aggressively against the application in his own case of the principles of the open-air treatment. It is useless to try and convince such a person that fresh air is his best friend. He knows by experience that it is not; whatever its theoretical merits may be, to him it means undue loss of heat and consequent physical miseries. If he is ever to be convinced it must be by way of his skin. When that is properly educated and rationally clothed, draughts will cease to have any terror for him. Until then, he will remain a convinced and militant obstacle to progress, inflicting overheated and polluted atmospheres upon every company in which he may find himself. Considering that, thanks to the demon of tuberculosis, the vast majority are flannel-wearers, what possible chance under existing conditions has the true gospel?

If this demon is to be cheated, the community must be purged of this preposterous heresy. The skin must be trained by cold baths and exposure to other cold influences adequately to perform its contractile duties. Underclothing must be in consonance with physiological principles; that is, it must be warm, absorbent and ventilated. Nature did not intend us to live in hot houses, neither did she impose clothing upon us. If we would live healthfully and allow our children to develop normally we must revert to natural conditions so far as the exigencies of civilised life will permit us. The necessity for pure air is gaining recognition, but it will not carry us far until we have recognised the equal if not greater necessity for rational and physiological treatment of the skin. Linen, cotton, and silk under-garments can be made, and are made, so as to imprison air in their meshes Such garments are every whit as warm as flannel, to which they are superior in their possession of those absorptive and ventilating properties of which flannel is so conspicuously devoid.

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A CLINICAL LECTURE ON ACHYLIA.

Delivered at the Polyclinic.

By ROBERT HUTCHISON, M.D., F.R.C.P.,
Physician to the London Hospital.

GENTLEMEN,—I wish to talk this afternoon about "achylia," or to give it its full title, *achylia gastrica*. It is a condition which has been curiously neglected in this country, although it has been very well recognised in America and on the Continent. Why it has been neglected here it would be difficult to say, unless it be that in this country we do not make use of the stomach-tube for the diagnosis of intestinal and gastric disorders so much as is done elsewhere—perhaps not so much as we ought—and achylia is a condition which can be recognised only when the stomach-tube is used.

It may be necessary to define more fully what I mean by the term "achylia." Achylia is a condition in which there is a great diminution of the gastric juice, certainly as regards its hydrochloric acid, and usually also as regards its ferments, so that when you come to examine a sample of the contents you may find them to be scarcely acid at all. Further, the stomach is completely empty between meals, and its mucous membrane is abnormally vulnerable. These are the chief features of the disorder as laid down by Einhorn, who used the term first in the year 1892, although the condition had been recognised as existing before he labelled it with that name.

Pathology.

With regard to the pathology of achylia, there are two possibilities: one is that it is a purely functional disorder. That is a view that is held by some; they say it is a disorder of innervation, and that just as there may be a functional increase

of hydrochloric acid in the gastric juice, so there is no reason why there should not be functional diminution. In favour of that view is the fact that unquestionably some cases of achylia are recovered from. I have never seen a case of recovery myself, but several have been described where a patient had had achylia for a time and yet afterwards the gastric juice had reappeared. That is most easily explained on the assumption that the disappearance of the gastric juice was due to a temporary or functional disorder. The other view, the one which is perhaps more widely held, is that achylia is due to organic change, which takes the form of an atrophic gastritis, or, in other words, an advanced condition of chronic inflammation of the mucous membrane of the stomach. In favour of that view there is clear histological evidence, for in several cases which have been investigated after death there has been found chronic inflammation of the mucosa and atrophy of the secreting tubules. The question most in dispute, however, is whether atrophic gastritis covers all the cases, and whether the supposed functional cases in which the gastric juice reappears after a time are not due simply to a slight degree of gastritis, which disappears with the restitution of the mucous membrane to its normal condition.

Relation of Achylia to Pernicious Anæmia.

Before speaking of the symptoms and signs of achylia I want to discuss briefly with you its relation to pernicious anæmia. There can be no question that if you examine the stomach functions in patients who are suffering from pernicious anæmia, you will find that they exhibit absence of gastric juice. In other words, pernicious anæmia is characterised by achylia, and the same is true of other cachectic states. Patients with advanced carcinoma, for instance, even carcinoma of other organs than the stomach, may exhibit achylia. The question is, What is the relationship of the two conditions. It was suggested by Austin Flint about 1860 that pernicious anæmia might be the result of atrophy of the gastric mucous membrane, and about ten years later Dr. Samuel Fenwick actually proved by microscopical examination in a case of pernicious anæmia that atrophy of the stomach mucous membrane did exist. After that the belief was held by many that the essential element in the pathology of pernicious anæmia was this atrophy of the mucous membrane of the stomach, which atrophy produced both the achylia and the blood-state. It was believed, in fact, that pernicious anæmia was brought about by the patient being unable to digest albuminous food, owing to the absence of gastric juice. We have, of course, advanced a long way since those days, and we now know that pernicious anæmia is not produced in that way, also that atrophy of the mucous membrane of the stomach does not always result in pernicious anæmia. For if you examine the blood of many patients who have had achylia for years you will find that it may be perfectly normal. On the other hand, I am not prepared to say that there is not something in the old view as well. For instance, I have seen at least two patients who presented the group of symptoms which we label pernicious anæmia, and who exhibited also achylia, and in whom, so far as one could gather from the history, the symptoms of achylia had preceded by some years the development of pernicious anæmia. I am not certain, therefore, that to suffer from achylia does not predispose a patient to the development of pernicious anæmia, or in other words, that there is not some relationship such as was outlined by the older writers, though not so frequently or invariably as they supposed. Undoubtedly achylia may be present for many years without pernicious anæmia resulting, but it may very well be proved that in some way, not yet understood, absence of hydrochloric acid and of gastric juice opens the way to pernicious anæmia, because we know, or at least many hold, that pernicious anæmia is due ultimately to some gastro-intestinal disorder, and it is possible that the absence of hydrochloric acid, which is the normal antiseptic of the intestinal tract, may predispose to the development of that particular form of disorder. However, I do not propose to take up more time over this point, which is after all an academic one, and shall now pass on to speak of the symptoms by which you may recognise achylia gastrica and the physical signs which are associated with it.

Symptoms of Achylia.

As regards the symptoms, you may divide the cases into three groups. First, *those which show no symptoms.* There are patients who have suffered from achylia, so far as one can tell, for years, and in whom the condition was discovered

quite accidentally, and who have never had any symptoms directly referable to the achylia at all. That is a rather surprising fact, and it illustrates again the great reserve power of the alimentary canal, how, even when one organ, such as the stomach, is not producing its digestive secretion, other organs, such as the pancreas, may so perfectly replace the functions of the stomach that there is no actual or discernible disorder of digestion to be detected. How numerous those cases are we do not know, of course, because we are not in the habit of examining people's stomach contents indiscriminately when they seem to be in perfect health.

The second group are those in which there are gastric symptoms of some sort, *i. e. dyspepsia.* Now, there is no one kind of dyspepsia which is absolutely characteristic of achylia. You cannot say in connection with any group of symptoms of gastric discomfort or disorder that they indicate absence of gastric juice. But one symptom which is always suspicious when it occurs, and which I have seen in these patients more often than any other, is persistent regurgitation of food, a regurgitation which goes on painlessly, and which has often persisted for years. I do not say that this is a symptom which is by any means pathognomonic of achylia, but it is one which is always a little suggestive and suspicious when it does occur. In addition to that the patient may complain of loss of appetite, although that is not by any means constant. It is surprising that this symptom is not *more* constant, because, as you know, there is believed to be a very definite relation between the subjective sensation which we call appetite and the secretion of gastric juice, and in most cases where the gastric juice is diminished, such as when there is gastric catarrh present, the appetite falls off. Sometimes, again, the patient complains of such symptoms as fulness and feelings of distension, symptoms which may be associated with many forms of gastric disorder, and which therefore are not of any value for diagnostic purposes.

The third and most important group of cases are those in which *the symptoms are entirely intestinal.* These are the cases the real nature of which is constantly overlooked, and which cannot be recognised and treated properly unless you are on the look-out for them and make an examination of the stomach contents. It is probable that in about one fifth of the cases the only symptom of achylia gastrica is intestinal disorder, which expresses itself usually in the form of more or less chronic or recurrent diarrhœa. I will describe an example just to bring before you, by an actual case, the sort of thing I mean. A man came to Out-patients' at the London Hospital complaining of rather severe attacks of diarrhœa, which kept coming on at frequent intervals and constantly troubling him, and at the time of his visit had troubled him for some months. There was nothing very special about the diarrhœa; he simply passed several liquid motions in the morning, with a scalding feeling at the time. He was a working man and looked perfectly robust and healthy. There was nothing in his habits to account for the diarrhœa, he had had no previous illness, and was a total abstainer from alcohol. I tried in his case various methods of treatment, empirically, including various astringents, and as he got no relief I at last took him into the hospital and started to investigate him more thoroughly. I began by giving him a test-meal, and as a result of that I found that he was not producing any gastric juice at all. The test was repeated on two or three occasions and always with the same result. I then began to give him hydrochloric acid, and adapted his diet to the absence of gastric juice. As a result of that treatment his diarrhœa speedily ceased, and although at different dates afterwards he had recurrences of the diarrhœa, yet the attacks were always arrested by his taking hydrochloric acid again. I have seen several similar cases since then, and they illustrate the fact that achylia may show itself by purely intestinal symptoms, for the patient of whom I spoke made no complaint of anything which you could describe as dyspepsia at all: his appetite was good, and as far as he knew he always digested his food properly; the only condition which troubled him was the recurrent diarrhœa. To such diarrhœa the term "gastric diarrhœa" is suitably applied.

Gastric Diarrhœa.

You may ask why the fact that the stomach is not producing gastric juice should cause a person to suffer from diarrhœa, and that is not so easily answered. There are two explanations which suggest themselves: one is that the diarrhœa is due to increased intestinal putrefaction from the absence of hydrochloric acid, hydrochloric acid being the normal antiseptic of the alimentary

canal, as I have already said. In favour of that view is the fact that patients who suffer from gastric diarrhœa exhibit other signs of intestinal putrefaction. They do not stand well a diet which putrefies easily; for instance they will tell you that they cannot take meat. Two of my patients with achylia adopted vegetarianism voluntarily without knowing that there was anything wrong in the stomach. Further, patients with achylia pass large quantities of indican in the urine, which is a sign of increased intestinal putrefaction.

The other explanation of gastric diarrhœa which suggests itself is that it may be due simply to mechanical irritation of the intestine by the undissolved food. The function of the stomach, as you know, is to reduce the solid diet to a liquid form, the subsequent stages of digestion being carried on lower down in the bowel, and if the food is not reduced to chyme but passes into the bowel in solid masses, it may irritate the intestine mechanically and so cause diarrhœa. I have observed more than once after the operation of gastro-enterostomy—it is one of the remote sequelæ of that operation which comes before the physician and which the surgeon does not hear anything about—that chronic diarrhœa has developed, and I think it has been proved by giving such patients bismuth meals that the food gets down into the cæcum sooner than it should. After an efficient gastro-enterostomy the food may reach the cæcum in one and a half hours instead of about five hours as is normal. There I think the diarrhœa is due to the direct irritation caused by the food passing through the stomach into the bowel before there is time for it to become dissolved, and the trouble can often be rectified by seeing that the food is thoroughly minced before the patient gets it.

The exact explanation of how gastric diarrhœa is brought about, however, does not interest you nor me as clinicians. The important thing for us is to recognise the fact that chronic diarrhœa is often due to absence of gastric juice, however it be that the absence of gastric juice produces the diarrhœa.

These being the symptoms, either none at all, or dyspeptic, or, in one fifth of the cases, purely intestinal, I pass now to speak of the signs of achylia, because if the symptoms are so uncertain and equivocal it is necessary that we should have some signs to guide us if we are to make a diagnosis.

Signs of Achylia.

I would say at once that to ordinary physical examination cases of achylia may present no signs at all. It is only by means of the stomach-tube that the condition can be recognised with certainty. When you give a test meal to such a patient and then pass the tube you obtain certain striking and characteristic results. In the first place you may be unable to get away any contents at all, or at all events very little. The stomach seems to be empty much sooner than it should be. But here arises an interesting question: is it really empty? It has been asserted by some, and I think rightly, that the absence of gastric juice leaves the food in such a viscid condition, a consistence more or less like porridge, that you cannot get anything to pass up the tube and so the stomach seems empty. But that will not cover the whole case. There is a tendency for the stomach to empty itself too soon as well, for if you wash out the stomach after a test meal you will get away much less material than in an ordinary case. Now we know that the mechanism which closes the pylorus is the development of free hydrochloric acid in the stomach, and in cases in which there is an excess of hydrochloric acid there may be spasm of the pylorus. Hence it is perfectly understandable that where there is a diminution of hydrochloric acid the pylorus may not be sufficiently locked up; it opens too soon, and allows the food to pass before the normal time. Further, the matter has been put to the test of experiment. Patients who suffer from achylia have been given first of all an ordinary test meal, and the amount left in the stomach after a given time has been estimated and found to be very small. Then there has been given along with the test meal a quantity of hydrochloric acid, and after a similar lapse of time the amount left in the stomach is found to be greater, showing that the addition of hydrochloric acid to the test meal prevents the stomach emptying itself so soon. I think, therefore, that this sign is due partly to the difficulty of getting away the contents because they are so thick, and partly to the stomach becoming emptied of its contents sooner than is normal

The second thing about the stomach contents that will strike you is that the food is there in lumps just as it was swallowed. The fragments are not swollen up nor partly dissolved. In the third place there is no free hydrochloric acid present. That does not surprise you; but if you estimate the total acidity you will find it to be very low indeed. In some cases it is so low as scarcely to turn blue litmus paper red. If you examine for the ferments you will find in a considerable number of cases that there is no renin present. The pepsin is diminished also, but not so constantly as the rennin. These characteristics, then, distinguish achylia: the comparative absence of the stomach contents altogether, the fact that they are scarcely acid at all to litmus paper, that they contain no free hydrochloric acid, that rennin is frequently absent, and that the contents exhibit no sign of having been acted upon by gastric juice. By means of these signs it should be possible to give a certain decision in almost every case.

Treatment.

With regard to treatment, the first question is the choice of a diet. Here, I think, the most important thing to do is to present the diet to the patient in such a form that it will not need to be reduced to a semi-fluid condition by the stomach. The function of the stomach being, as I have said, to convert the solid food into semi-solid, you will present the diet with that part already done. It need not be in the form of slops, but animal food finely minced, and vegetable food passed through a sieve. Further, you will recommend very thorough chewing. The chief food which the stomach digests is meat, and I think it is advisable to restrict meat in cases of achylia, particularly when diarrhœa is present. One of my patients who suffers from complete achylia, an intelligent man, who observes his own case carefully, found he could not stand starchy food well unless he took with it some form of diastase. That is a difficulty which other patients with achylia have complained of, and it is one which it is difficult to understand, because you would expect that where there was no acid the diastase of the saliva would have an opportunity of acting on the stomach contents and converting the starch. Many of these patients also cannot stand milk unless it be first peptonised. So a partially predigested diet containing little meat and in which the food has been reduced to a pulpy condition is the best all round.

We have next to consider the use of drugs in the treatment of achylia. If there is such a thing as "functional" achylia, *i. e.* a diminution or disappearance of gastric juice from some temporary disturbance of innervation, then one would think it might be possible by giving agents which we know stimulate gastric secretion to produce good results. With this object bitters have been given, especially the extract of Condurango. I do not think, however, that you will find this plan very successful. One has therefore to fall back upon attempts to replace the gastric juice artificially. You can do this by the administration of hydrochloric acid and ferments. Particularly in the cases with diarrhœa I think hydrochloric acid is extremely useful, but in all cases it is worth a trial. What suits some patients better is "acidol" (betain-hydrochloride), which liberates hydrochloric acid in the stomach. It can be procured in $7\frac{1}{2}$-grain tablets, of which the patient takes one or two after each meal. I have one patient who has been taking them for more than a year. After he had been taking them for some time I tried how he would get on with a suitable diet alone. But he wrote and said he could not get on without the acidol; he said he was not so comfortable and got "fermentation" in the intestine as soon as he left it off. I do not think it is so important to replace the ferments. I do not myself believe much in artificial ferments, such as pepsin, given by the mouth, and you will remember that the pancreas is capable of doing all the digestive work which is required. Probably the reason why hydrochloric acid is of so much more advantage than the ferments is, that hydrochloric acid, as you will remember from the work of Pawlow, is the normal stimulant which leads to the production of pancreatic juice, and if hydrochloric acid is absent the pancreatic juice is apt not to be produced in sufficient quantity. If you give hydrochloric acid you supply a stimulus to the pancreatic secretion, which then does the digestive work. I have found some advantage, however, from ingluvin, which is a ferment prepared from the gizzards of fowls. I have usually given it with hydrochloric acid.

To conclude, the chief points I want to impress upon you are, first, that achylia is a real condition, secondly, that it is not sufficiently recognised in this country, although it is not very infrequent, and thirdly, that it is a disorder which may manifest itself by intestinal symptoms only, and should be thought of especially in all cases of chronic diarrhœa which you cannot explain on other grounds.

January 4th, 1909.

WITH MR. BLAND-SUTTON AT THE MIDDLESEX HOSPITAL.

IV.

GENTLEMEN,—To-day I propose to consider with you the subject of cancer as it affects the alimentary canal, because it shows in striking contrast the rapidity with which cancer sometimes destroys life when it occurs in the alimentary canal as compared with the breast. It is, in fact, an impressive illustration of the effects of environment in connection with this disease. The whole of this subject is extremely interesting and important, because more than half the total number of cases of cancer which attack the body arise in connection with the alimentary canal. If you take independent organs of the body and compare the stomach in this respect with the breast and the uterus, these three situations prove to be the most common in which cancer arises. For my own part I think the order in which they stand in this respect is that cancer of the breast is the commonest, that cancer of the uterus and cancer of the stomach are about of equal frequency. But some people who have paid great attention to the statistics of the matter are of opinion that cancer of the stomach is commoner than is cancer of the breast, or of the uterus. That is striking enough, but if you take the whole of the alimentary tract, from the œsophagus to the anus, then this important point comes out: that more than half the cases of cancer which arise in the body generally arise in connection with the alimentary tract. The distribution of cancer in the alimentary canal is important, for it is common in the œsophagus, extremely frequent in the stomach, but rare in the small intestine. It is uncommon at the ileo-cæcal valve, but very common in the large bowel and rectum. Of course all this is of great interest to those who are working at the origin of cancer, because the frequency with which cancer attacks the large intestine is in favour of those who believe that cancer is due to a micro-parasite, probably something which is taken in either with food or with water. Indeed I feel so strongly on this matter myself in reflecting on this subject of cancer of the large intestine that for many years I have avoided eating all sorts of uncooked vegetables. Much as I love salad with my chicken or my cheese I do not touch it. I do not wish to prejudice you against the delights of the table, but I think these facts are worth bearing in mind. However clean your celery may appear to be, if you care to carefully pull aside the stalks immediately around the centre of the celery, which is about the only part worth eating, you will see that there remains some dirt which has not been washed away. In regard to the preparation of uncooked vegetables for the table I think it well to draw your attention to the remarks of a Frenchman visiting England. He said, "You English are a remarkable people; you live well and like to eat vegetables, but it is the only country in the world where the scullery-maid is trusted to prepare the vegetables." There is much truth in this remark.

Some years ago, when I made almost daily visits to the Zoological Gardens for the purpose of making post-mortem examinations of the bodies of animals dying in the menagerie, the extreme frequency with which their viscera were invaded with parasitic worms was a matter of great surprise to me, and they were often present in uncountable numbers. On contemplating such things one felt deeply thankful that most of the food eaten by mankind is cooked. We know full well that living on partially cooked or "under-done" meat carries penalties in the way of Entozoa, as the intestinal parasitic worms are called.

Some years ago I operated on a patient for appendicitis, and in the early stages of the disease she suffered from persistent diarrhœa. To relieve this she had taken for several days a quantity of raw pounded beef on the advice of her doctor. As a consequence she suffered severely from an acute infection of beef tape-worms (*Tænia medio-canellata*). Events of this kind make us agree with Metchnikoff that "Entozoa may serve as gates of entry for the hypothetical parasites of carcinoma." There are many channels by which minute but not invisible Invertebrata gain an entrance into the alimentary canal. Food and water are essential to life, and it is undeniable that they are the frequent purveyors of disease, and that minute animal parasites are our daily messmates and table companions, and whose presence we sublimely ignore.

To return to uncooked vegetables. One of the most striking observations relating to this matter we owe to Behla concerning the town of Luckau in Germany. This town contained 5000 inhabitants,

and the majority were engaged in agriculture. Between the years 1875–1898 the death-rate from cancer was very high, and numbered 1 out of 25–30 for the entire town. The cancer cases, however, did not arise in all parts of the town. More than half the population inhabited its central portion and the eastern suburb, where the houses were similar in size and arrangements, and as a rule damp. The soil of these parts of the town was moist and lay low. A large ditch encircled the central town and the eastern suburb; the cancer followed closely the course of this ditch, which contained foul and stagnant water, and in which the people watered and washed their vegetables, many of which were eaten raw. The western suburb was sandy, dry and elevated, and remote from the ditch. In this part of the town there was not a single death from cancer during the period investigated by Behla.

In Behla's opinion the probable source of infection was in the uncooked vegetables.

Behla believes that it is experimentally proved that cancer is due to a micro-parasite, and that it is a vegetable organism which enters the blood through skin or mucous membrane; its spores penerate epithelial cells and cause enlargement of their nuclei and asymmetrical mitoses, and later they produce proliferative irritation. Finally a sporangium is found within the cell, or close to it in the infected tissues. As these parasites have not yet been cultivated experimentally the cause of cancer still remains, like that of smallpox, in the domain of hypothesis, although, be it remembered, there are several very infectious diseases of which the parasite has not been detected, much less cultivated, and yet the infectivity of the disease is beyond dispute.

Let me draw your attention to the unequal distribution of cancer in the alimentary canal.

The œsophagus.—Compared with the other parts of the alimentary canal the mucous membrane of the gullet is not very vulnerable to cancer. It is more common in advanced life, and may be described as rare before the fortieth year. Any part of the œsophageal mucous membrane may be attacked, but it is more common in the upper than in the lower half. Carcinoma of the gastric end of the gullet is uncommon. This I thought might be due to some difference in the method of tabulating the cases, for it seemed possible that in some statistical tables cancer at this end of the œsophagus might be tabulated as belonging to the stomach. This is correct, but even including such cases it is true that cancer around the terminal orifice of the gullet is uncommon.

Nothing is known of the early stages of this disease, for it produces no symptoms until the œsophagus is narrowed and hinders deglutition. As soon as the disease is revealed in this way its course is very rapid. The patients die from inanition and exhaustion due to obstruction to the passage of food, pleurisy and septic pneumonia. Death also occurs from hæmorrhage when the cancerous ulceration opens up large vessels in the mediastinum, especially the aorta.

The stomach.—According to some statistical researches cancer arises in the stomach more frequently than in any other organ, and it is especially common in the parts lying along the lesser curvature and in the vicinity of the pylorus. This is a noteworthy fact, because this area may be described as the chief district frequented by ulcers. This is a matter of great interest, because many surgeons hold the opinion very strongly that cancer is prone to attack chronic gastric ulcers and the scars which they leave in healing. This is only in accord with the habits of cancer in other parts of the body, namely, it attacks by preference chronically diseased organs and tissues. In this respect a chronic gastric ulcer may be considered as a precancerous condition. Gastric cancer runs, as a rule, a rapid course. Life is rarely prolonged beyond twelve months from the time the disease is first recognised. This quickly fatal course, especially when the pylorus is implicated, is due largely to the obstruction offered to the escape of food through the pylorus. Death may be due to slow starvation, hæmorrhage, perforation into the general peritoneal cavity, septic pleurisy from perforation of the diaphragm, general dissemination and thrombosis.

The small intestine.—Primary cancer of the small intestine is uncommon; perhaps the usual spot in which it arises is in the second part of the duodenum, at or near the bile papilla. The next point is at the ileo-cacal valve, but here the disease is uncommon, especially if care be taken to distinguish between cancer arising in connection with the valve and cancer originating in the mucous membrane of the cæcum.

The large intestine and rectum.—Primary

cancer of the large intestine is a common disease, and the liability of its mucous membrane to become cancerous increases from the cæcum to the rectum. Thus cancer is thrice as common in the terminal two feet of the large gut (sigmoid flexure and rectum) than in the remainder of the colon, and if care be taken to discriminate between hyperplastic tuberculous disease of the bowel and cancer the disproportion will be found even greater.

Of the initial and early stages of cancer of the colon we know nothing: the disease has no special symptomatology, and causes no pain until it has so narrowed the lumen of the gut that the intestinal contents (fæces) are hindered from passing; then the patient suffers from intestinal obstruction, and seeks advice, and, in many instances, when the cancer reaches this stage unless surgical help is quickly forthcoming the patient will succumb from perforation of the cæcum, or the colon above the structure.

The character of the disease in the early stages is unknown, for it grows unsuspected and unrecognised, and in an absolutely painless and unsuspected manner until the lumen of the colon is too narrow to allow fæces to pass, then pain arises as a sequence, and even then the disease remains undetected often for many months. In the colon cancer appears in its final stages in two forms. Thus it may be massive and exuberant, the cancerous mass blocking up the bowel. The other form is the more insidious, for the cancerous growth may resemble the small contracting nodule such as I showed in relation with the breast. When cancer attacks the colon it spreads around it in a circular manner, so that in the contracting form it will so narrow its lumen that in the advanced stages the colon will appear as if a piece of cord had been tied tightly around it, as these specimens on the table prove.

I have long held the opinion that in these extreme contracting forms of cancer of the gut we have, as I endeavoured to explain to you in connection with the small contracting forms of cancer in the breast, examples of a primary cancer undergoing spontaneous cure. Here, of course, the cure is almost worse than the disease, for this very complete cicatricial contraction of the primary growth leads to the very condition which renders colic cancer such a rapidly fatal disease, namely, obstruction of the intestine.

When cancer arises in the rectum we find an additional element of danger, because the cancerous mass is in close proximity to the bladder and the ureters. In the late stages of the disease these organs become involved, and, apart from the extreme misery produced by the formation of urinary fistulæ, the patient's life is terminated by septic invasion of the urinary system, which leads to death by uræmia.

The comparison of cancer of the breast and cancer of the gastro-intestinal tract in relation to its life-destroying properties is extremely instructive. As we saw in a former lecture, cancer, when it attacks the breast, runs an insidious course, and only a small proportion of its victims die from the direct result of the primary disease, whereas when cancer attacks the stomach and large intestine primarily it so profoundly interferes either with alimentation or with the escape of the gross excrementitious products of the body that life soon becomes impossible. Thus from the practical side of our work it is incumbent on all to keep themselves thoroughly informed of these variations in the clinical behaviour of cancer in these different situations.

From the scientific point of view the liability, or better, the vulnerability of the various sections of the alimentary canal to cancer is most instructive in relation to its ætiology. For example, those parts of the digestive tube are most prone to become cancerous where food makes the longest sojourn. The œsophagus is merely a conduit or food duct. The stomach is a churn, and in this hollow viscus there is much mixing and motion of the ingested material, and therefore prolonged and close contact. The digested mass is hurried through the small bowel into the cæcum, where it is retained for many hours and is ordinarily deprived of the main part of its fluid. That the undigested mass known as fæces is retained for many hours, and in some ill-conditioned individuals for days, in the descending colon and sigmoid is an undeniable fact.

This retention would be favourable for the invasion of the epithelial covering of the gut by an infective agent.

January 4th, 1909.

THE SURGERY OF RECTAL STENOSES.

A Post-graduate Lecture delivered at the London School of Clinical Medicine.

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GENTLEMEN,—Like the rest of the alimentary canal the rectum is, as you are aware, liable to stenoses of various degrees, and arising out of divers conditions at nearly every period of life, and the treatment of these stenoses is, unfortunately, far from satisfactory. In this respect the rectum is to some extent comparable to the œsophagus, since, like that tube, its position precludes the reconstitution of its lumen after the removal of any considerable portion of its length, except in the immediate vicinity of the anus.

The discomfort and misery induced by such a condition as stenosis render it one of the most terrible afflictions, and one which we, as medical men, are only rarely able to cure.

I do not propose to discuss that form of stenosis which arises through pressure from without, for this is, as a rule, a temporary obstruction pathologically unconnected with the rectum, and belongs to the department of pelvic surgery.

The variety of rectal stenosis which arises earliest in life is that which results developmentally from mal-position or mal-union of the proctodæum with the hind gut in the fœtus, and may be brought about in two ways; for either the natural absorption of the septum between these two structures may be arrested, leaving the resulting passage unduly narrow, or a total failure to absorb may compel the surgeon to have recourse to puncture and dilatation, or to prolonged dissection, the stenosis unavoidably resulting having thus a traumatic as well as a developmental origin. Fortunately these forms are distinctly rare, and, except in the second instance, their presence may not be suspected till later on in life, owing to the natural softness of the excreta in the infant. They may, however, be discovered long before puberty, and in the absence of other causes their true origin will probably be recognised. Their special characteristic is the absence of thickening, and the presence either of simple narrowing, or of a hymen-like ridge representing the remains of the partially obliterated septum.

Next in order we may consider those stenoses which result from traumatic causes. These may be either self-inflicted injuries, or the direct result of operations performed upon the rectum.

Some years ago I was asked to see a young man who had a very serious stricture of the rectum; it was in the second portion, and would not admit anything larger than a No. 7 bougie, and was surrounded by a very large amount of inflammatory thickening. It was certainly not the result of syphilis, and on inquiry I learnt that a year previously he had suffered from a severe attack of enteric fever, and during his convalescence obstinate constipation had necessitated the constant use of enemata, which were administered by an unskilled attendant; these enemata resulted at times in sharp hæmorrhage. After his recovery he still continued the enemata himself, always employing a very long and rigid tube, and I was forced to the conclusion that the stenosis was the result of repeated abrasions of the mucus membrane. I have since that time seen an almost identical case, and Allingham has recorded another in an elderly lady.

The stenoses which result from operation are, I need hardly remind you, of the highest importance to us as medical men. Fortunately, better methods have greatly reduced the occurrence of these unfortunate accidents; they are, however, still commoner than is generally supposed. Two forms of operation stand out in my mind as being particularly dangerous in this respect: the first of these, which is fortunately becoming obsolete, is that for the cure of hæmorrhoids by the use of the clamp and cautery; and the second is that which bears the well-known name of Whitehead, and which, as you know, is a partial excision of the rectum for diffuse hæmorrhoidal conditions.

The first of these operations, which was at one time extensively practised, results unavoidably in large sloughing areas, and ultimately in the development of much cicatricial tissue in the submucous coat of the bowel. In expressing an adverse opinion on Mr. Whitehead's operation, it must be remembered that Mr. Whitehead has laid great stress on the observance of the rules which he laid down for its performance, and in his hands

it has probably given excellent results. It has, however, frequently been applied by other and possibly less skilful operators to conditions for which it was never intended, and to areas of mucous membrane other than those for which it was designed; and I have myself seen several cases in the course of some years of out-patient practice which, as the result possibly of the imperfect application of the operation, possibly of some subsequent accident, have developed as firm and intractable stenosis as would have followed an extensive gummatous ulceration. In some hundreds of cases I have never found it necessary to employ this method, and, indeed, I am inclined to class it among certain other operations as being of the nature of a "surgical gamble," that is to say, among operations which, if they are not successful, leave the patient in a much worse condition than he was in previously.

The stenosis occasionally supervening on Whitehead's operation originates in several different ways, all of which should be remembered by those contemplating its performance. Firstly, the sutures used may be too rapidly absorbed; they may be tied too tightly, and so cut out too soon; they may be too few in number, and be placed too near the edge of the mucous membrane. Secondly, too large an area of submucous coat may be denuded, and thus the tension on the bowel brought down may be too great for these sutures. Thirdly, imperfectly controlled oozing from this submucous surface may separate it from the mucous membrane, and result in the formation of pus; and here it must be remembered that when an excessive amount of submucous tissue has been bared, the attempt to cover the raw surface by drawing down the cut margin of the mucous membrane to the anal verge, is bound to result in the formation of a potential cavity in the form of a circumferential tunnel, which, unless very carefully obliterated by pressure, is certain to fill up and suppurate.

The next class of stenosis to be considered is that which results directly from inflammatory diseases, and amongst these syphilis undoubtedly accounts for by far the greater number of strictures, and probably for those of greatest severity.

Let us consider the other rarer causes first, and return to syphilitic stenosis later, taking it as the type of stricture most commonly requiring our attention.

Dysentery is a much less common cause of stenosis than syphilis, and is usually less severe in its effects; but where the ulceration resulting from this disease is followed, or, indeed, accompanied by cicatrisation, the stricture is commonly very serious, owing to the height at which it at times occurs and to the extensive area involved. And this extent fully counterbalances the fact that the depth of the cicatrix is distinctly less than in the case of syphilis. Further, owing to the multiplicity of ulcerated areas the resulting cicatrices are more prone to form irregular masses, bands and ridges. For the above reasons the treatment by dilatation, which is often applicable to specific, is often impossible in the case of dysenteric stenosis.

Tuberculous disease of the rectum, although resulting at times in extensive ulceration, is seldom the cause of extensive stenosis; indeed, a stenosis from this disease is a comparatively rare occurrence, and I can only call to mind one or two cases in which it has led to any serious stricture. The reason is probably to be found in the fact that tuberculous ulceration is commonly confined to the mucous membrane, and does not invade the submucous or muscular tissues to any great extent.

Another cause of stenosis, and one, perhaps, more commonly at work than is generally recognised, is gonorrhœal infection; more commonly seen in women, in whom it is derived as a secondary infection from the vagina, it results in a very virulent form of acute proctitis and periproctitis, with ulceration and sloughing which lead to a marked degree of stenosis, which is no doubt often supposed to be syphilitic.

The ulceration produced by the presence of stercoral masses in the rectum is said at times to lead to stenosis, but although I have on many occasions removed from the rectums of elderly patients stercoliths of some size I have seldom met with ulceration, and I have never seen a stenosis in these cases.

To return to syphilis as a cause of stenosis, there is, perhaps, no condition of the rectum so worthy of our attention; for although cancer is a more terrible disease, its period of activity is comparatively short, whereas the stenosis of syphilis occurs in young people with all their life before them, and consequently their prospects, their health, and their happiness may be hopelessly wrecked if means cannot be found to arrest the

disease before it has done what no amount of surgical skill can undo.

Of all non-malignant stenoses of the rectum, that which results from syphilitic ulceration is the most rapidly established, the most insidious in its onset, the deepest in its cicatrix, and the most intractable to treatment. Its commonest situation is the third part of the rectum just above the internal sphincter; and the question is often asked, why should it choose this particular site? The following reasons may explain this propensity to some extent. Firstly, syphilis is a disease especially affecting fibrous tissue. Now it is to this point in the rectum that the pelvic fascia descends to split into its recto-vesical and anal layers; and here also we find the attachment of the levator ani, between the posterior fibres of which lies the fibrous connection between the rectum and the coccyx. Again, it is at this point that the chronic congestion consequent upon the presence of hæmorrhoids occurs, for here is one of the meeting-points of the systemic and splanchnic circulation, and sluggishness of the venous current is at all times a predisposing cause of the formation of fibrous tissue. Local irritation, as in the case of the tongue, œsophagus, etc., may have some influence, and possibly the starting-point of a gumma may be found in the presence of abrasions or stercoral ulcers.

There are certain points with regard to this form of stenosis which are of interest. Thus, it is not uncommonly the only lesion present, no other sign of specific taint being evident. Unlike the stenosis of dysentery, it is usually established without the pain, ulceration, and discharge—the earliest sign of trouble often being an increasing difficulty in evacuating the bowels. Its progress is thus extremely insidious, and by the time it is established the period of grace has passed and it will be found that the usual syphilitic remedies are powerless to resolve the dense mass of fibrous tissue of which it is composed.

The special predisposition of women to specific stenosis of the rectum is believed by Hutchinson to be due to the damage done to that organ during labour by the passage of a large head. The chief objection to this would seem to be, that sloughing of the rectum, when it occurs from this cause, is usually seen at a higher level, viz. opposite the sacral promontory; and secondly, that many cases of stenosis which are attributed to syphilis are seen in women who have never been pregnant.

The last class to be considered is that in which the stenosis is malignant. What connection, if any, does this condition bear to the non-malignant type? In many cases there is no doubt a very close connection, and in the case of syphilis this connection has long been recognised in the tongue especially. Examples of this are seen in the conversion of the smoker's patch and other varieties of syphilitic leucoplakia into epithelioma: and where the irritative cause is simple, of granular cervix uteri, chronic balano-posthitis in old men, or the bald anal and pudendal skin commonly seen in the case of long-standing pruritus in women, becoming gradually malignant. It is therefore reasonable to suppose that chronic irritation of a syphilitic stenosis of the rectum with its ulcerations and fistulæ would commonly result in cancer, and that therefore cancer of the rectum should be more common in women than in men. This, however, is not the case, for according to the statistics of Allingham, of all cases in St. Mark's Hospital five sevenths occurred in men and only two sevenths in women.

In spite of the above remarks there is no doubt that cancer of the rectum may originate *de novo*, never having been preceded by any pathological condition.

Now, as regards the diagnosis of commencing stenosis the great difficulty in getting these cases in time is the attitude of the patients themselves: in the early stages, stenosis of the rectum, whether specific or malignant, is a perfectly painless condition, and consequently the trouble progresses insidiously in either case, till the disease is fully established, before the patient's attention is directed to one of three signs: either there is a sense of fulness in the rectum and of something to be expelled, or there is gradually increasing constipation, or there will be the passage of a little blood. The last of these is unfortunately the only one of the three which commonly appeals to a man; women neglect all of them, for they commonly suffer from "bearing-down" pains; constipation they appear to regard rather as a natural destiny of their sex, while they are so accustomed to the sight of blood that it appears to many women to be all the same whether it comes from the front or back passage; if it comes from the latter they will always provide you with a diagnosis of piles.

While on this subject I cannot help impressing upon you a very important matter which has come under my notice on many occasions. That women, and even men, should, without a second thought, attribute rectal hæmorrhage to the presence of hæmorrhoids, as if no other disease could exist here, is perhaps not to be wondered at in an age when the public goes about seeking to be deceived by every Christian Scientist, charlatan and quack who may bray loud enough; but that members of our profession should tamely accept the diagnosis of hæmorrhoids from the mouths of their patients, and should prescribe for them without ever taking the trouble to make a rectal examination, is little less than criminal; and yet in the past few years I have come across many such cases in out-patient practice, and some of these cases proved to be not hæmorrhoids but carcinoma. It seems almost unnecessary to say that hæmorrhage from the bowel, although more commonly due to hæmorrhoids than any other condition, should invariably be an indication for a rectal examination, for in this way alone can the diagnosis be made certain, and mistakes fatal not only to life but to reputation be avoided.

I propose, however, to deal only with the question of simple stenosis here, since that of malignant stricture is in itself a very large subject. In considering the curability or incurability of stenosis arising from any of the causes I have mentioned several factors must be discussed. First of all, the level of the trouble is of importance, since stenoses in the upper part of the rectum are less easily reached for dilatation, and their dilatation is fraught with greater risk than when they are situated just within the anus. Secondly, their extent must be considered; the length of some of these strictures is remarkable, and their calibre frequently varies at different points, the tortuous nature of the lumen rendering the passage of sounds difficult or impossible. Thirdly, we must ascertain whether or no active ulceration is present; and lastly—a point often difficult to diagnose—whether there is more than one stricture present.

With regard to this last point, I may say that double strictures are rare; but they do occur, as pointed out by Emil Ries, who has reported two cases, but as a rule it is not possible to diagnose them before operation.

The height and length of rectal stenoses depend very much on their cause, and, as will be readily understood, extensive superficial ulceration, such as results from dysentery or tubercle, are more liable to produce long strictures which are less densely fibrous than the localised, deeply situated lesions of tertiary syphilis. It may be said that as a general rule stenoses which result from syphilis, and, indeed, most others, when once they are fully established are perfectly incurable by drugs, and only very rarely curable by any operative procedure: the ulcerations produced by dysentery and tubercle are in themselves so chronic that even if they are eventually overcome the submucous fibrosis has progressed to such an extent as to preclude the possibility of cure. Syphilitic stenosis, on the other hand, may, and often does, occur without any precedent ulceration, and herein lies its insidious nature; in the absence of pain and bleeding attention is not directed to the trouble, and it is only when the fibro-plastic mass formed begins to undergo contraction that the lesion is discovered. At this stage it can no longer be resolved, no matter how largely iodides may be employed, and, unless operation is feasible the condition is perfectly incurable. If, however, the gummatous deposit is discovered while yet the mass is immature, it may be resolved just as easily as elsewhere in the body.

Certain secondary changes are at times seen in these cases of stenosis: it is, of course, well known that enormous dilation above the stricture may occur, and result in the formation of stercoral ulcers; these at times lead to the formation of fistulous tracks of a serious type, which in their turn tend to increase the amount of fibrosis about the rectum. Then, again, pruritus of the anal region is of common occurrence, and may be almost maddening in its intensity. Another secondary effect which I have noticed frequently, and which is yet seldom mentioned in the literature of stenosis, is the tendency of the stricture to affect the muscular action of the sphincters, even when at quite a high level. The anus, as the result either of partial disuse, or, when the stricture is low down, of nerve implication, becomes weak and patulous, being to some extent robbed of its duty; thus flatus and liquid fæces pass from time to time involuntarily, and the patient is much distressed. Again, in those cases in which the stenosis is at a higher level than the internal sphincter the com-

pression undergone by the veins of the rectum results in the development of hæmorrhoids.

Lastly, one must not forget the possible mental effect of stenosis on patients who are possibly highly strung, especially if the stenosis be specific in origin. In these patients, already affected by syphilophobia to a greater or lesser degree, the constant worry of a rectal stenosis is frequently the starting-point of morbid mental changes, which, being favoured by the resulting chronic anæmia and ill-health, often lead on to suicidal mania.

A very important consideration is that of the ultimate prognosis in rectal stenosis. What is the prospect of such cases progressing to malignant disease in later life?

I believe that such change depends to a great extent on several factors. Thus, heredity must always very largely influence the eventual result. Syphilis bears, as is well known, some very definite relationship to malignant disease; and therefore one would be less surprised at malignant change in a stenosis of specific than in one of dysenteric or tuberculous origin. At the same time we know that many cases of specific stenosis remain non-malignant throughout life. The fact probably is, that ulceration has much to do with the result. Those stenoses which are of such small calibre that ulceration due to stercoral abrasion is almost unavoidable, in which bougies have frequently to be resorted to, and in which enemata are in constant request are in a constant condition of irritation, and are thus liable to alter their character and become malignant; whereas in those in which the stenosis is not so marked, in which bougies are seldom necessary and in which ulceration has perhaps never occurred, there is no special reason why malignant change should manifest itself other than the possible predisposition of specific lesions already mentioned. I am inclined to think, therefore, that *ulcerated* stenoses of specific origin are, like chronic ulcerated leucomata of the tongue in syphilitics, extremely dangerous lesions, but that simple stenoses without ulceration do not necessarily tend to carcinomatous change.

In considering the treatment of rectal stenosis one is almost inclined to despair of any progress in this department of surgery, for, although in other respects the surgery of this region has advanced, we are still, in the face of stenosis, little beyond where we stood twenty years ago. Little has been done in the way of prophylaxis, and we are still compelled in most cases to fall back on dilatation by bougies—at best a very painful and unsatisfactory method, and in unskilled hands often dangerous.

In the first place treatment will depend on whether ulceration is or is not present: to attempt to dilate a stricture of the rectum in the presence of ulceration is not only useless, it is bad surgery; the ulceration is aggravated, the inflammation is stimulated, and the fibrosis is increased, the stenosis being rendered more intractable than ever. Nor is this all; in the case of stercoral ulcers the result of forcible dilatation may be to set up a septic pylephlebitis; in that of dysentery or tubercle the result may be liver abscess or tuberculous lesions elsewhere. Where the passage of blood, pus, or mucus indicates that ulceration is present, dilatation is definitely contra-indicated; the ulceration must first be dealt with, and this, I need hardly say, is a very difficult matter. If the ulceration is slight, and the stenosis low down, it may be possible to treat it by local applications in the shape of injections or suppositories, but in the case of long strictures, or of those situated in the upper part of the rectum, such a method is impossible in the ordinary way.

In other parts of the body the first indication in the treatment of ulceration is to cleanse the ulcer, the second is to remove any cause of irritation, and the third, to promote healing by local applications; in the case of the rectum it is unreasonable to expect the accomplishment of the first two of these indications so long as the continuity of the bowel is maintained. The repeated passage of fæcal masses over the ulcerated surface is compatible neither with cleanliness nor with rest. In the urethra the passage of sounds, catheters and urine through an inflamed and irritable stricture is bad surgery, and we tide over the difficulty by perinæal section, diverting the stream of urine till such time as the inflammation shall have subsided. In the rectum, however, we hesitate to apply the same logical reasoning, owing to the greater magnitude of the risk and the possible unpleasantness of the procedure; and yet this risk is nowadays very much less than it was, and better means of cleanliness have been devised. It is, to my mind, quite unreasonable to allow a patient to suffer the agony of ulceration, with its tenesmus, frequent evacua-

tions, loss of sleep, anorexia, and general misery and ill-health, and to expect that with all this such ulceration is going to resolve one day and allow the use of dilating bougies. So far from doing so, it will so increase the firmness of the stricture that dilatation can no longer be carried out at all.

I would therefore urge the performance of colotomy as a temporary measure in all cases of ulceration of the rectum, *before* the occurrence of stenosis, as a prophylactic measure. It will cleanse the rectum, give physiological rest to the ulcers, it will abolish the spasm of the sphincters, it will give relief from pain and promote sleep, and it will permit of lavage and the application of remedies to the part in a rational manner from above the seat of trouble. As regards the ulceration of tubercle and dysentery, its extent is often unknown, and it may even involve the cæcum; it is, however, in the rectum that its stenotic effects are most marked, and most difficult to deal with, and therefore our efforts should be directed especially to this region.

Stenosis unaccompanied by ulceration is quite a different matter. Here, as a rule, there is no acutely painful condition, but simply one of discomfort and ill-health, and as regards the treatment there are several alternatives: they are, simple dilatation, proctotomy with dilatation, Péan's rectoplastic operation, exclusion by simple anastomosis, the anastomosis known as sigmoideo-proctostomy, complete excision, resolution by drugs, and, lastly, permanent colotomy. Let us consider these alternatives briefly.

Dilatation is, in my experience, rarely satisfactory, and is only permissible where the stenosis is near the anus, of slight extent, and not tortuous. It can, as a rule, only be satisfactorily applied by skilled hands, and many patients (especially women) are too nervous to attempt it themselves, to say nothing of the danger of self-inflicted injury, and the fact that it is a painful method which has to be kept up throughout life.

External linear proctotomy must be mentioned to be justly condemned: that it is dangerous is shown by the statistics of Lachowski and Sonnenburg, who state that 15 per cent. of patients died as the result of operation, presumably from sepsis, while most of the survivors had recurrence of the stenosis within a year.

Internal linear proctotomy is less objectionable, as the incisions are not so deep, but for this very reason but little improvement is possible and more fibrosis is created.

Péan's operation is only suited to narrow stenoses such as those which result from the congenital abnormalities originally referred to; it is useless in cicatricial strictures. It is carried out much in the same manner as the operation of pyloroplasty, the stenosis being divided in the vertical axis of the bowel, and the wound being then converted into a transverse one. Needless to say the opportunities for its performance are not numerous, nor has it met with a great amount of success.

The next method to be mentioned is that of simple anastomosis, the sigmoid above the stricture being brought into contact with the rectum below it. This operation has been performed several times by Bacon, who reports twelve cases in which it has been successful. The anastomosis was effected by means of Murphy's button, one half of which was inserted through the abdomen, and the other half by the rectum. Quite apart from the fact that very few cases are suitable to this method, the stricture being of too great an extent, the large intestine, and especially the rectum, has always been considered a situation to which this button is unsuitable; at the same time it is the only feasible method of effecting an anastomosis so low down, unless the very much severer method to be described is adopted. This is the operation first described by Emil Ries, and known as sigmoideo-proctostomy, and which was later practised by Rotter and Kelly. Through a transverse incision in the perinæum the recto-vaginal or recto-urethral septum is split, and the dissection is carried right up to the pouch of Douglas. Through this gap the sigmoid is drawn down and divided. The upper end of the rectum is then closed by suture, and the lower cut end of the sigmoid is brought below the level of the stenosis and implanted into the anterior wall of the rectum above the sphincters. The operation is severe and difficult, and it has the disadvantage that the stenosed portion of the bowel, with its ulcerated surface, if such is present, is left *in situ*. Its success depends very largely on the length of the meso-sigmoid, and there is considerable probability of the bowel being again stenosed by the contraction of the tunnel through which it has been drawn, just as one at times sees in the case of colotomy wounds, in which the bowel

passing through the abdominal wall is often tightly gripped by the tissues. Ries' case seems to have relapsed after a period of seven years. Rotter and Kelly claim to have had some success.

Theoretically, the ideal method of dealing with stenosis is that of complete excision, and provided the stenosis is low down, and not of great extent, is worth trying. It is, of course, essential that the sphincters be preserved, and that no great amount of tension be placed upon the sutures. It is not, however, an easy operation, owing to the free hæmorrhage which takes place from the cut surface of the fibrous material; nor are the cases to which it is applicable numerous. Schede and Mickulicz have reported twenty-three cases, with one death and about 40 per cent. of cures. It closely approximates to Whitehead's operation in this, that if the stitches give way from suppuration or excessive tension the condition is rendered as bad, if not worse, than before.

I have left till now the consideration of how far it may be possible to bring about a resolution of the tissues of which a stenosis is composed by the use of drugs, as it is only within comparatively recent times that such a possibility has been contemplated. You are probably aware that in thiosinamine the property has been said to exist of causing a resorption of fibrous cicatrices such as those of Dupuytren's contracture, of severe burns, and of specific stenosis of the gullet. In 1901 Hebra and Juliusberg recommended its use in scleroderma. Many writers have testified to the efficacy of this drug, but of their cases I will only mention such as appear remarkable or which have some relative bearing upon the stenosis we are considering. In 1904 Hartz recorded a case of pyloric stenosis in which resolution occurred as the result of twenty-three injections of 1 c.c. of an alcoholic 15 per cent. solution of thiosinamine, two injections per week being given. By a similar method Halasz effected in the same year the resolution of a simple stenosis of the œsophagus. In this year Merck, of Darmstadt, produced a combination of thiosinamine and sodium salicylate which he termed "fibrolysin," the addition of the latter drug being made for the purpose of preventing the precipitation of the former at low temperatures from aqueous solutions, and in the year 1906 Pollack succeeded with this substance in resolving a stenosis of the œsophagus of eight years' standing in six weeks, while Schourp appears to have been equally successful with it in cases of urethral stricture. Wesselberg has had success with this drug in œsophageal stenosis, as has also Bass, and Frank and Lang have spoken of its efficacy in urethral strictures.

The drug is injected into the gluteal muscles every second or third day, and there seems to be considerable unanimity of opinion that twenty-five to fifty injections are required to bring about an improvement. I have not been able to find any record of its application to rectal stenosis, but it seems to me, in view of the reported results in stenosis of other mucous tracks, an investigation in this direction might be worth attempting.

Lastly, there remains permanent colotomy, a measure, it is true, only to be adopted when all else fails, and yet, although it has been much abused, one which is capable of bringing the greatest comfort and relief to those who agree to submit to it.

The general practice among surgeons at the present time is still to advocate the performance of iliac colotomy, and in some cases this operation may have the best claims to your notice. In 1906 I advocated the adoption of transverse colotomy in a paper communicated to the Clinical Society as the operation of election in these cases, and since that time I have found no reason to alter my opinion Briefly, the advantages which this operation offers over the older method are as follows:

The transverse position of the colon here does away to a great extent with the effect of gravity upon the contents of the bowel, and thus minimises the risk of constant extravasation and prolapse of the mucous membrane. The fact that many cancers of the upper part of the rectum are found to have caused infiltration and thickening of the meso-sigmoid, renders it at times difficult to bring the sigmoid into the wound without the exercise of an inconvenient, and possibly dangerous amount of tension; for the same reason it is not always possible to bring the sigmoid sufficiently near the middle line to utilise the fibres of the rectus muscle as a sphincter, and unless this can be done, the linea alba being a weak spot, this sphincteric action is lost. The iliac region in stout patients is often occupied by a fold of abdominal fat, which renders the firm application of a belt and pad a matter of some difficulty, while in those who

are emaciated the prominence of the iliac spine, covered by thin and stretched skin, offers a favourable situation for the occurrence of sloughing, to which the frequent contact with fæcal matter contributes. The epigastric region, supported as it is by the prominence of the ribs, the presence of the liver, and the firm scaffolding of the lineæ transversæ of the rectus muscle, which is here thick and well reinforced by two layers of sheath in place of only one, enables the patient to apply such pressure to a belt as would cause great discomfort at the iliac region; it also permits of the belt being supported by braces over the shoulders, and of the formation of a very efficient sphincter from the rectus muscle; this position is moreover much more accessible to the patient for the purposes of bodily cleanliness. Lastly, in cases which are not malignant, and where the short-circuiting of the intestine may possibly enable the surgeon to bring about the closure of a fæcal fistula, the reconstitution of the bowel after such closure is much simpler of accomplishment in the case of the transverse than in that of the sigmoid colon.

January 4th, 1909.

'The Pros and Cons of Vivisection,' is the title of a book by Dr. Charles Richet, of Paris, with a preface by Professor W. D. Halliburton, published by Messrs. Duckworth & Co., of 3, Henrietta Street, Covent Garden. Professor Richet has given particular attention to the study of the psychological side of physiology, and his views on pain will be read as coming from one who is specially fitted to deal with this and other mental phenomena. We find Professor Halliburton's preface so excellent that we do not hesitate to quote freely from his well-expressed teaching. He says that if there is one piece of evidence more than another which was given before the Royal Commission that deserves rescue from the oblivion of a blue book, it is that given by Lord Justice Fletcher Moulton. He begins by taking the case of a ship infected with plague, and infested also with rats, the carriers of plague. The ship enters port. Would it be preferable to kill the rats, and so prevent them and the disease from entering the port, and causing untold disaster there, or staying one's hand because the slaughter of the rats would be a painful proceeding? The captain who gives orders for the destruction of the rats inflicts pain and death on them in order to prevent greater pain and more widespread death elsewhere. The captain who says "Spare the rats" is guilty of the criminal act of causing the death of many innocent human beings. So it is with the anti-vivisectionists: they see only the pain inflicted, and do not heed the pain prevented. On this score they are in a sense logical when they call Lord Lister a brute, although he, of all men living at the present time, has been the means of preventing the greatest amount of suffering. They see only the pain which he deliberately inflicted on a few rats and rabbits; they cannot see, or refuse to see, the measureless amount of misery he has prevented. In another place the Lord Justice points out that the pain inflicted in all the laboratories of the country put together during a year is infinitesimal compared to that which is inflicted every day in the slaughter of animals for food; to that which ignorant farm labourers inflict without anæsthetics, in spaying animals by thousands in order that beef and mutton may be tenderer or have a more pleasant flavour to the consumer; to that inflicted by sportsmen when their victims, imperfectly shot, die a lingering death; to that which women thoughtlessly allow in order that they may have ospreys in their hats and furs upon their backs. So far as the satisfaction of appetite, the pandering to the so-called sportsman's instincts, or the gratification of vanity are concerned, these things may go on. The average anti-vivisectionist disregards them, or at least makes no effort to prevent them. The only kind of pain which stirs his feelings, and meets with his opprobrium, and enables him to indulge in his favourite epithets, is the one justifiable bit of pain in the whole world—a pain inflicted with the noblest of all objects, and by the most humane of all men (for so the medical profession admittedly is), the object, namely, of preventing future pain, which otherwise would encompass the world of life.

Corrigendum.—On page 166 of The Clinical Journal of December 23rd, 1908, the illustration printed above the words Fig. 1 should have appeared over the words Fig. 2 on page 167 of The Clinical Journal, December 23rd, 1908, and the illustration over the words Fig. 2 should have been placed over Fig. 1.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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CHRONIC PNEUMONIA.

A Clinical Lecture.

By JOHN FAWCETT, M.D.,
Physician to Guy's Hospital.

I HAVE only to-day commenced my duties in "Clinical," and consequently I have had no opportunity of selecting a case from that ward. As the subject of my lecture I propose to take one of those rare cases of diffuse fibrosis of the lung which may be included under the title of this lecture.

To start with, it seems desirable to indicate to you what is meant by "chronic pneumonia," or, as it is variously named, "chronic interstitial pneumonia," "pulmonary cirrhosis," or "fibroid lung." These names are given to a *clinical* group of cases of long duration, owning different causes, in which the replacement of the pulmonary tissue by fibrous tissue is sufficiently extensive to give rise to a definite set of physical signs and symptoms. "Fibroid phthisis" is a name which has in past times been used as an equivalent title of this group, but it is quite wrong according to our present-day pathology, because "pulmonary phthisis" nowadays implies an invasion of the lung by the tubercle bacillus, with its accompanying changes. This name should on no account be employed in describing conditions which are not a part of a pulmonary tuberculosis.

Thus the case of which I shall now relate to you particulars is not one of chronic phthisis, but, as I take it to be, a chronic pneumonia of doubtful origin.

Alfred E—, æt. 50 years, was first admitted under my care on March 21st, 1900, with profuse hæmoptysis. He was a painter, and had been at work as usual when, about 5.30 p.m., he suddenly felt a choking sensation in his throat, which was im-

mediately followed by a rush of bright-red blood into his mouth.

His previous medical history is given in the report as follows: Five years before he had an attack of "bronchitis and pleurisy," and since then he had been subject to a cough. In 1898 he expectorated a quantity of bright-red blood, and had continued to do so in small amount at infrequent intervals since that time.

The patient was well nourished; his face was somewhat blue; his fingers were "clubbed," and he was distinctly short of breath. He was emphysematous, and the cardiac impulse could neither be seen nor felt. On admission no minute examination of the lungs was made, but rhonchi were audible over both lungs. Expectoration was abundant.

March 26th, 1900.—Blood was absent from the sputum for the first time. Loud râles were heard over both bases, more especially on the right side.

April 6th and 7th.—A little blood mixed with the sputum.

The temperature for the first five days varied between 101° and 103° F., but for the rest of the patient's stay in hospital, with the exception of one or two slight rises, it remained at the normal level.

Neither tubercle bacilli nor any elastic fibres were found in the sputum on two examinations.

The patient was re-admitted under me on May 10th, 1902, as he had on the previous day brought up "a teacupful" of bright blood. For the past two years he had been at work, and although some cough with expectoration had persisted, and he had, at rare intervals, expectorated a little blood, yet, on the whole, his progress had been satisfactory.

His symptoms were similar to those on the first occasion. He had lost no weight, nor had he suffered from any symptoms such as night sweats and diarrhœa, which so frequently accompany a chronic tuberculous affection of the lungs.

Two days after admission the sputum was still bright red and frothy, but a few days later the hæmoptysis had entirely ceased. Tubercle bacilli were again sought for without success. At first there was some pyrexia, lasting for five days, the highest temperature recorded being 102° F. He was discharged on May 23rd, 1902.

After this date the man attended at my "Out-patients" more or less regularly. He was able to continue at his work, except when his cough became more severe, but he had no sufficiently severe attack to necessitate his again being treated as an in-patient until October, 1908, when he came complaining of quite different symptoms, which proved to be due to a dilated stomach, the result of a carcinomatous stricture of the pylorus, which ultimately led to his death.

I have so far said little about the physical signs, because I wished you to obtain a general idea of the features of the man's illness, particularly in relation to—

(1) Its duration of, probably, ten years at least.

(2) The attacks of hæmoptysis, accompanied by definite rise of temperature of five days' duration, and followed by extension and alteration of the physical signs over the right lung.

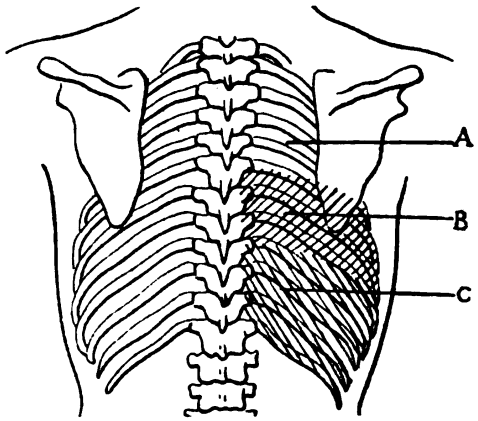

Diagram of posterior aspect of lung. A. Some few rhonchi and râles. B. Impaired resonance with bronchial breathing, bronchophony and pectoriloquy. C. Impaired resonance and deficient vesicular murmur.

(3) The chronic cough, dyspnœa, cyanosis, and clubbing of the fingers.

(4) The absence of general constitutional symptoms, and of involvement of other organs.

(5) The absence of tubercle bacilli from the sputum.

As regards the physical signs they were those of bronchitis and emphysema, but, in addition, over the lower right lobe there were others which are depicted in the diagram.

Thus, in addition to the bronchitis and emphysema, there were in the second attack signs which indicated a process of consolidation over the lower part of the right lung, with probably some thickening of the pleura.

From the amount of expectoration it was thought that dilatation of the bronchial tubes also existed.

Taking all the facts of the case into consideration, especially in regard to its duration and the absence of tubercle bacilli from the sputum, it was thought that the most likely diagnosis was a chronic pneumonia with bronchiectasis, and this was ultimately found to be the case.

At the autopsy old fibrous adhesions of the pleura were found all over the right lung, attaching it to the diaphragm and to the pericardium. The lower right lobe was contracted and fibroid. The left lung was in a condition of hypostatic pneumonia. There were no tuberculous foci anywhere.

Ætiology.—In discussing the ætiology of a case like this one, I will first of all enumerate to you the conditions which are thought to precede the development of chronic interstitial pneumonia.

They are lobar pneumonia, broncho-pneumonia, pleurisy, obstruction or occlusion of a bronchus, syphilis, and pneumonoconiosis. Syphilis of the lung and pneumonoconiosis are nowadays, and quite rightly, treated separately in the text-books, just as is tuberculous pulmonary disease. Nothing, therefore, more clearly indicates to you that this group is merely a clinical one, and in process of time it will, no doubt, become still further depleted as the ultimate causes of the various forms of chronic pneumonia are recognised. In this particular case there was no question of syphilis or of any "occupation" cause being the deciding factor in the production of the disease. Therefore we have to fall back upon the other pathological states referred to. First of all let us take (1) *acute lobar pneumonia.* Now, in the case of Alfred E—, although the history is indefinite, yet it seemed improbable that his condition was the consequence of an attack of acute pneumonia. Rather did the history suggest some subacute recurring inflammatory lesion of the lung and its coverings, which had gradually resulted in a diffuse pulmonary fibrosis. It is generally accepted that a lobar pneumonia may subsequently develop into a "chronic pneumonia," although all writers agree that this event is one occurring very rarely indeed. A patient who recovers from an acute pneumonia generally does so completely. In a debilitated patient, whether due to serious disease of other organs, or in a drunkard, resolution may be hindered, but if, though tardy, improvement is gradually progressive, the case cannot properly be called a chronic pneumonia.

Charcot's case is, perhaps, the one most frequently quoted in support of the statement that chronic pneumonia may be the result of an acute pneumonia. The patient in question died four months after the onset of an acute attack, and at the autopsy the lung was the seat of a grey fibroid change throughout, while the opposite lung, though enlarged, was free from disease. Addison again described conditions which he spoke of as "uniform albuminous degeneration" and "grey induration" as sequences of acute pneumonia.

Other writers have reported like cases and like pathological conditions. Thus, even if one admits the possibility of the sequence of events just referred to, yet it is only with the proviso that it is very rare. On the whole the clinical history in these cases is very unlike that of the acute classical variety.

We will, therefore, now consider what may be called Group 2, *subacute or primary interstitial pneumonia.*

This group is one to which Dr. P. Kidd, among others, drew attention some years ago, and he described it by the name of "subacute indurative pneumonia." If we examine the records of the cases reported by Charcot, Coupland and others, we see that both the onset, the course, and the symptoms differ materially from those of the acute variety, and it appears almost impossible to deny, in these cases, the pre-existence of chronic disease upon which a more or less acute process had supervened.

It seems, therefore, very reasonable to suppose that most of the cases of this class of chronic pneumonia are a distinct variety, and are due to a slowly progressive subacute fibrous change in the lung rather than a chronic pneumonia following an attack of acute lobar pneumonia.

Dr. Kidd quotes two cases, and the one I have related to you to-day belongs, I think, to this group. In support of this statement is the long duration of the case, the gradual development of the physical signs of consolidation of a persistent kind, accompanied by periods of pyrexia, but exacerbations which, all of you who have carefully followed the details of the case will agree, have no resemblance whatever to an attack of acute lobar pneumonia.

To impress the characteristic features of this group upon you I should like to relate to you

another case which was under the care of Sir Cooper Perry, and which is, I think, without much doubt a case of primary subacute pneumonia.

William K—, æt 45 years, was seized with a sudden pain in the left side twelve weeks before admission into hospital, followed by a cough and increasing dyspnœa. There was no history of syphilis nor any evidence of it, past or present. The man was a compositor, but stated that his work was neither dusty nor did it necessitate working in a badly ventilated room. He was a non-smoker, drank no spirits and very little beer. The patient was very definite with regard to the onset of his illness, and the doctor in charge of him said that at no time, in the three months previous to admission, had he shown any symptoms of an acute illness, like lobar pneumonia. There was some impairment of note at both bases, and T.V.F. was deficient; the respiratory murmur and voice-sounds were deficient at the bases, and no adventitious sounds were audible. There was no organic disease elsewhere, and no tubercle bacilli were found in the sputum after frequent examinations. At the end of the first month in hospital the percussion note at the right base had become resonant, while that over the left side was still impaired. The percussion note beneath the left clavicle was also impaired. During the second month in hospital cough was more marked and sputum more abundant in quantity. In the course of the third month the percussion note became still more impaired, but at no time was there any typical bronchial breathing, the inspiratory murmur being harsh, as was also the expiratory murmur, and in addition unduly prolonged. The temperature frequently rose two or three degrees above normal at night, and the respiration rate gradually increased to 40–50 per minute. Death occurred six months from the onset of the illness, being preceded by an attack of acute bronchitis and pleurisy.

At the autopsy recent pleurisy was found, with some old adhesions on both sides. The upper lobe of the left lung and the upper part of the lower lobe were slate grey in colour, firm and tough, cutting with difficulty on account of the fibrous tissue with which it was permeated. The lower half of the lower lobe was dark in colour, heavy and friable. In the right lung the appearance was similar, but the change did not seem to be so far advanced. Some of the tubes in the upper lobes were slightly dilated. There were no thick septa, no cavities, and no evidence of a tuberculous invasion. On histological examination a great increase in the fibrous tissue of the lung was found, involving the walls of the alveoli. The alveoli contained exudation products which were undergoing conversion into fibrous tissue. In some of the alveoli blood-vessels were seen springing from the walls, while in others the vessels were obliterated by the newly formed fibrous tissue.

If this patient had had an attack of acute pneumonia the case would have been regarded as a typical example of a chronic pneumonia secondary to an acute one, but the doctor who attended him throughout the period before he came into hospital corroborated the patient's statement as to the gradual onset of the illness and of its indefinite signs in the early stages.

The features of this variety of chronic pneumonia are different to those of acute pneumonia. The fever is of an irregular type, and there is no sudden crisis. The physical signs are of slower development, less well defined, and do not disappear. The constitutional effects, at any rate in the early stages, are less marked.

The cases of this group related by Heitler succumbed in periods varying from five weeks to nine months. It is, however, quite likely that, as in the case of Alfred E—, the disease may become partially arrested and persist as a chronic pulmonary lesion.

Group 3. "*Pleurogenous*" *pneumonia.*—Some cases of pulmonary fibrosis are thought to originate from a pleurisy, a great overgrowth of fibrous tissue taking place with subsequent extension into the lung. Whether this be actually the case or not, whether, for instance, as Wilson Fox thinks probable, there is an accompanying pneumonia, yet the fact remains that there is a group of cases in which pathologically the pleura is very greatly thickened, and the fibrous tissue is seen spreading into the lung from the pleura in all directions. Recently, while going through the Guy's Hospital post-mortem records for fifty years, Dr. Rippmann and I found forty-six cases of chronic pneumonia or pulmonary cirrhosis which exhibited changes sufficiently marked to give rise to physical signs. During this period 21,317 autopsies were made: this shows the rarity of the disease. We did not,

according to the definition given at the beginning of this lecture, include any cases which might have had a syphilitic, a tuberculous, or an "occupation" origin. Twelve of these forty-six cases may be said to belong to the "pleurogenous" group, in which the condition of the pleura is quite different to that in any other group. The pleura is described, for example, as being "obliterated and half an inch thick," as "an eighth of an inch thick, with trabeculæ passing into the lung," and so on.

Indeed, the extension into, and the invasion of, the lung from the pleura is so well defined in this group of cases that it seems hardly possible to doubt the causal connection between the two, although it may be impossible to prove it.

Now, neither in the case of Alfred E— nor in Sir Cooper Perry's case was the condition of the pleura anything like what is found in this group of cases, a group to which most of the cases occurring in adults belong, and from which many of the descriptions of "fibroid lung," both as regards its clinical aspects and post-mortem appearances, are derived. The lung in the pleurogenous group is frequently very much reduced in size, shrunken to a third or less of the size of the opposite lung, and consisting of little more than fibrous tissue, which usually is in the form of dense white strands, branching in all directions, and having a gristle-like consistence when cut into. Amongst this tissue, thickened dilated bronchi are frequently seen, the pulmonary tissue which is left being of a dark, blackish colour. Clinically, too, this group is representative of the extreme effects that ensue in the development of a fibroid lung—a result which is readily comprehensible after gaining a mental picture of what has just been shortly described. Thus the corresponding side of the thorax is deformed and sunken, the shoulder is perhaps depressed, and even the spinal column may be curved laterally. On the other hand, the opposite lung is large and emphysematous, and free from disease, while the heart is pulled over towards the affected side.

The following case, that of Jas. E. A—, æt. 35 years, who was under the care of Dr. Newton Pitt in 1904, is an example of this group. As so often happens in chronic cases the history is very imperfect, but it appears that the man had had an illness some five years before, followed by a persistent cough, and that he had been twice previously an in-patient in the hospital, where a diagnosis of "fibroid lung" had been made. There was shrinking and flattening of the left side of the thorax, and the fingers were "clubbed." Death ensued as the result of an attack of acute bronchitis, and at the autopsy the left pleura was found to be an eighth of an inch thick and adherent throughout, with septa of fibrous tissue traversing the lung. The lung was shrunken to a third of the size of the right lung, or even more than this, and the lung tissue was replaced by cartilage-like fibrous tissue, while the tubes were dilated. The right lung was compensatorily hypertrophied.

GROUP 4. *Following broncho-pneumonia.*—In this connection I will first of all shortly describe to you the case of a boy, æt. 10 years, upon whom I performed an autopsy eight years ago. He had an attack of measles and whooping-cough six years previously. During his last year of life he had four sharp attacks of pneumonia, for all of which he was an in-patient in the hospital where he died. In the first of the four attacks of pneumonia, in September, 1898, there were signs of general bronchitis, with dulness and bronchial breathing at the base of the left lung. In the three later attacks, in March, August and September, 1899, there were signs of broncho-pneumonia; in the intervals between these attacks some bronchitic signs persisted. With each acute attack there was a rise of temperature up to 101°–103° F., falling with the subsidence of the attack to about normal.

At the autopsy both lungs were found to be very tough and of a reddish grey colour, the change being more marked in the left lung. A diffuse peribronchial and perivascular fibrosis was present, and there was also alveolar fibrosis and thickening of the pleura.

Now, there are several features exemplified by this case, which are more or less characteristic of the group.

Firstly, broncho-pneumonia, preceded by measles and whooping-cough, is the most frequent forerunner of pulmonary fibrosis in early life. Clark, Hadley and Chaplin state that the great preponderance of this cause over all others is only met with in early life, and this is borne out by our Guy's series of cases. In the forty-six Guy's cases fourteen followed broncho-pneumonia, and of these eight died within the first five years of life, and of

the remainder only two were over twelve years of age, one being seventeen years and the other twenty-seven years old.

Secondly, in many of these cases the degree of shrinkage of the lung is very much less than in the "pleurogenous" group; the pleura, although commonly affected, is not so to the same extent, and consequently the physical signs may be, and often are, far more indefinite.

Lastly, inasmuch as relapsing broncho-pneumonia is practically non-existent in adults, that is, apart from the forms due to a specific cause such as tubercle, or from irritation by particles of dust, no difficulty arises in explaining the absence of broncho-pneumonia as a factor in adult life.

GROUP 5. *Due to compression or occlusion of a bronchus.*—This cause is one to which little attention is paid in text-books except in relation to the production of bronchiectasis. Notwithstanding this we found seven cases in the forty-six from the Guy's records in which a definite fibrosis of the lung had developed. The bronchiectasis is, perhaps, the most striking naked-eye feature of the lung in these cases, and yet the physical signs are due to the fibrous change in the lungs rather than to the dilatation of the tubes.

From the number of cases above recorded this group is obviously one to be remembered, especially because of the frequent difficulty of diagnosing the cause of the pulmonary fibrosis. If we examine the seven Guy's cases, we find that three were due to the pressure of an aortic aneurysm and four of them to a "a malignant growth" obstructing the bronchus. These two conditions are the only common causes, although, of course, other mediastinal tumours which obstruct a bronchus may produce the same result.

Perhaps the most fertile source of failure to diagnose the cause of the fibrosis is where there is a small aneurysm of the commencement of the descending thoracic aorta, which, giving rise to no recognisable "pressure" signs or symptoms, is only diagnosed when it ruptures, or the discovery is made on the post-mortem table.

That the fibrosis is no mere pathological curiosity is evidenced by the descriptions given in the post-mortem reports; in all the seven cases the lower lobe was the one affected, and the seat of the disease is variously described as being "densely fibroid," or as being "infiltrated with fibrous tissue."

To take an example of one of the cases: Henry S—, æt. 36 years, who was in Guy's Hospital in 1901, gave a history of pain in the chest with recurring hæmoptysis of some six months' duration. He suffered great pain, was dyspnœic, the heart was displaced towards the shrunken side—the left—and below the level of the sixth rib there was dulness, with râles. The diagnosis of basal phthisis had been made, as tubercle bacilli were said to have been found in the sputum. Ultimately a small aneurysm of the descending thoracic aorta ruptured into the left bronchus. At the autopsy this bronchus was found to be compressed by the aneurysm. The lower lobe of the left lung was fibroid, and the bronchial tubes were large and prominent.

In another of the four cases, Frank K—, æt. 38 years, was in Guy's in 1902. The report of the case states that the man had pneumonia and pleurisy about seven months before his death. The physical signs present on admission were chiefly those of deficient movement, deficient vesicular murmur, and voice-sounds on the left side, and later dulness over the same area. Death occurred from rupture of an aneurysm into the left bronchus, a condition which was not diagnosed during life. At the autopsy the condition found was similar to that seen in the previous case, viz. a small aneurysm of the descending thoracic aorta. Despite the use of X rays the post-mortem facilities of hospitals are likely to continue to afford us the opportunity of seeing similar cases, for it is in just such cases that an X-ray examination may prove negative, and even the most wary physician be defeated.

Thus, in a case of chronic pneumonia or fibroid lung of doubtful causation, remember obstruction of a bronchus, and not only obstruction due to aneurysm, but that due to a malignant growth, for in some of the cases of mediastinal cancer the cancerous cachexia is extraordinarily little developed, although in these latter cases there is not so likely to be a complete absence of auxiliary factors to diagnosis as there is in the case of a small aneurysm, which is no bigger than, or not as large as, a walnut.

GROUP 6. *Of doubtful origin.*—There were eleven cases in the Guy's series in which the cause of the fibroid change was doubtful. There was no specific cause, such as syphilis or pneumonoconio-

sis, to which the cases could be attributed, nor were there any indications in the past history of the patient which would allow us to allocate these cases to any of the groups already dealt with.

This difficulty in determining the ætiology is, however, readily understood when we remember how chronic the disease may be, extending over a period of fifteen to twenty years, or even more, and how imperfect is the past history of many of the patients.

Possibly Group 2, viz. primary chronic or subacute interstitial pneumonia, may claim some; the post-mortem appearances lend some support to this view, but it is impossible to decide from pathological examination alone, because similar appearances by no means imply similar causes.

This group, then, brings us to the end of the various conditions which lead to chronic pneumonia, and we will now shortly consider some other features, as instanced, first of all, by the two cases to which I primarily drew your attention.

Duration and prognosis.—In the first case, that of Alfred E—, you may remember that he lived for some years after he first came under my care, and it is probable that the disease commenced some four or five years previously. In the end his death was due, not to the chronic pneumonia, but to a carcinoma of the stomach.

In the second case, that of William K—, who was under the care of Sir Cooper Perry, death occurred six months after the commencement of the illness.

Now these two cases afford examples of two of the prominent features of the disease, viz. the chronicity in the one case, and secondly, that in the subacute indurative pneumonic group, or in those which supervene upon an acute pneumonia, the duration of the disease is considerably less than in the other groups. In Heitler's cases, quoted by Dr. P. Kidd, death occurred at periods varying from five weeks to nine months.

In a considerable number of the cases, however, chronic interstitial pneumonia proves to be a disease of long duration, ten, fifteen, or even twenty years it may be. On the other hand if the disease develop in child life, or if the disease is extensive, especially if associated with much bronchiectasis, the duration of life is generally much less.

There is no doubt that chronic pneumonia tends to materially shorten life. Of the 46 patients in the Guy's series 35, or 76 per cent., died before reaching the age of fifty years, and 29, or 63 per cent., before forty years of age.

When we investigated the causes of death in the six patients who succumbed between sixty-one and seventy years of age, we found that two of them had died from "malignant disease" occluding a bronchus, while a third, æt. 70 years, succumbed to an accident. Therefore, the percentage of deaths at the earlier ages is even higher than I have stated.

Again, the fatality of the disease in children is shown by the fact that 12 cases, or 26 per cent., died within the first ten years of life.

There is another interesting feature of chronic pneumonia, viz. that it is more common in men than in women, and in the Guy's series of cases there were thirty-nine males to seven females, and both the cases I have related to you to-day were in men.

Symptoms and physical signs.—The most prominent symptoms in the case of Alfred E— (Case 1) were those which are commonly met with in the disease.

They are cough, dyspnœa and expectoration, varying, of course, with the extent of the disease, but in all cases more or less marked.

The man, Alfred E—, became very easily short of breath; on any exertion his face became congested. His fingers were clubbed—a frequent result in chronic cases. Hæmoptysis in large quantity was, you will remember, a prominent feature, as he coughed up considerable quantities of blood on two occasions and smaller amounts at other times. Bastian found that there had been hæmoptysis in more than half of the cases in his series, and in several of the Guy's series hæmoptysis had occurred.

Another important feature of the chronic cases is the absence of general constitutional symptoms. The man, Alfred E—, remained well-nourished up to the time at which the symptoms of carcinoma of the stomach showed themselves. Except in the acute exacerbations this patient had no fever, and in many, or most, of the cases of chronic pneumonia there is little or no pyrexia unless some complication arises such as septic absorption from ulceration in dilated bronchi.

The physical signs of the lesion differ greatly,

according to the form and the extent of the disease, being often very indefinite at first, and later, perhaps, becoming well marked. In the case of Alfred E— there was an alteration in the percussion note, but the signs of consolidation were not well developed until the second time of admission into the hospital; they were associated with the expectoration of considerable quantities of sputum at a time, and hence it was thought that dilatation of the tubes was present.

In the case of the second man, William K—, who died six months from the commencement of his illness, you may remember that the note on percussion remained impaired, but there was at no time any definite bronchial breathing, although at the autopsy the left lung in particular was very firm and tough from the formation of fibrous tissue.

On the other hand in the long-standing cases, with much shrinkage of the lung there is retraction of the affected side and deficient movement, with, perhaps, depression of the corresponding shoulder; the note on percussion is dull, or more or less impaired; the vesicular murmur is deficient or absent over some areas and high pitched or bronchial in character over others.

Variation, again, is the characteristic feature of the adventitious sounds; for example, medium or large râles are heard, or where cavities or considerable bronchiectasis exists the râles may have a more consonating character.

Vocal fremitus, too, differs according to the form of the disease, in some cases being increased where there is little thickening of the pleura, while in other cases it is diminished; it is seldom altogether absent, as in the case of pleurisy with effusion.

Complications and results.—I have already referred to hæmoptysis and bronchiectasis in the cases brought before you to-day. Other complications are well represented by those we met with in our Guy's series. The patients frequently succumbed to affections of the cardio-vascular system, the right side of the heart being dilated and hypertrophied, and in many cases was associated with evidence of tricuspid regurgitation, with systolic pulsation in the veins of the neck and other signs of a disturbed and impeded circulation.

In one case the strain thrown upon the tricuspid valve, from the long-standing circulatory obstruction in the lungs, had led to thickening of the tricuspid valve and stenosis; in other cases thickening, dilatation and atheroma of the pulmonary arteries were present.

The second class of diseases to which the patients succumbed were acute affections of the lung, viz. acute bronchitis or broncho-pneumonia, or, where bronchiectasis existed, a septic broncho-pneumonia and gangrene.

The third class included various causes, such as cerebral abscess in one of the bronchiectatic cases, and a pneumococcal suppurative meningitis in one of the cases of recurrent broncho-pneumonia.

Other complications were simple and purulent pericarditis, pleural effusion and empyæma, and lardaceous disease.

It is not uncommon, too, in some of these chronic pulmonary lesions to find various forms of septic arthritis appearing, and then subsiding again, only to return once more later on.

Diagnosis.—This may be easy or it may be exceedingly difficult. In the case of Alfred E— (No. 1), the diagnosis was difficult at first, because the physical signs were so indefinite, and even though no tubercle bacilli and no elastic tissue were found in the sputum, yet we could not be sure that we were not dealing with one of those cases of very chronic phthisis, in which the tubercle bacilli in the sputum were very few in numbers and so had escaped detection. However, later on, when after two years no bacilli could be found, when the signs were chiefly basal and there had been no constitutional disturbance, no diarrhœa, no sweating, and no evidence of infection of any other part of the body, we arrived at the conclusion that the condition was not due to a tuberculous lesion.

This case shows you how important it is to try and put everything together before coming to a decision; it is not a question of one or more pathognomonic signs or symptoms, but it is the case as a whole, its past history as well as present, which must be carefully considered before a diagnosis is reached.

I have thus incidentally dealt with one of the main difficulties of diagnosis—the distinction from a chronic tuberculous lesion.

Perhaps, next to chronic phthisis, a chronic pleural effusion is very likely to be mistaken for a fibroid lung. The difficulty of these cases of long-

standing effusion is strikingly brought to your notice from time to time in the post-mortem room, where a localised encysted collection of fluid, either serous or purulent, is found, and there is reason to think, both from the history of the case and from the pathological appearances of the wall of the cavity, that the effusion had been there for months or even years. There are specimens of such localised collections in our museum, one of which had certainly been there nine years. In most cases of pleural effusion the heart is displaced away from the side of the effusion, but in some of the chronic encysted collections a contraction of the affected side occurs, with consequent displacement of the lung towards the side of the effusion. Then exploratory puncture is the only means of procedure, and this is frequently required to be performed in more than one place before a decision is arrived at. It is sad, too, to relate—perhaps it is so depressing that I ought not to mention it—that sometimes, after negative exploratory punctures, and consequently a diagnosis of pulmonary cirrhosis, the post-mortem examination discloses a local collection of pus after all.

Compression or occlusion of a bronchus leading to fibroid lung I have already dealt with, and I pointed out to you what a fertile source of error in this connection is an aortic aneurysm, and that in cases of doubtful origin, both aneurysm and mediastinal tumour are to be borne in mind as possible causes of the chronic pneumonia.

Treatment.—Inasmuch as the disease we have been talking of is only a clinical entity, so there is no specific treatment. The two cases we have taken to-day are examples of this fact.

The treatment is one adapted largely to the relief of symptoms, of cough, hæmoptysis, and of drugs to lessen the bronchial secretion or diminish fœtor, of drugs to relieve the cardiac and vascular conditions. In other cases surgical treatment may be required as, for example, if an empyema develops.

Apart from this, the treatment resolves itself into taking care that, when the acute stages have passed away, the patient is placed in as favourable conditions as possible for the maintenance of good general health, with plenty of fresh air and suitable food, and, when possible, spending the spring and winter in a warmer and more equable climate than England, such as on the Riviera, in Egypt, or in islands like the Canaries.

January 11th, 1909.

WITH MR. BLAND-SUTTON AT MIDDLESEX HOSPITAL.

V.

GENTLEMEN,—To-day I propose to consider with you the subject of villous disease or villous papillomata of the bladder. About a fortnight ago a woman, æt. 66 years, was sent into Bird Ward to see me, and she brought in her hand demonstrative evidence of her disease in the shape of a bottle filled with bloody urine. This at once showed that she had some disease either of her bladder, ureter, or the kidneys. The next and most obvious thing to do was to examine the urine and see if it contained anything which would suggest the cause of this dirty urine, and also to be sure that its colour was really due to blood. The urine was put into a urine glass and examined. It was of the colour of porter and contained a large amount of blood-corpuscles, but we found no evidence of epithelial cells. The next obvious thing to do was to admit the patient, and in due course we proceeded to examine the interior of her bladder. The proper way to do this, either in the case of a man or of a woman, when you think there is any serious disease of the bladder, is by means of the cystoscope. The old-fashioned way of examining the bladder of females, when we wished to know if there was anything wrong inside, whether a tumour or a hairpin, or a calculus, or a bodkin, used to be to dilate the urethra and put your finger in. It was a rough and ready way, but one which was very useful. When the urine is blood-stained the cystoscope will not light up the bladder, and you may wash out the blood-stained fluid, and often the more you wash it out the more the bleeding, as a rule, goes on. Therefore, to examine this woman's bladder by the cystoscope was out of the question, so I dilated the urethra and put in my finger, expecting to find a mass of carcinoma. But instead of that we were able to feel two small tufts of what is known as villous disease, or papilloma. Sometimes when these villi are not bleeding it is possible to gently fill the bladder with water and watch them by means of the cystoscope. When this is done skilfully they appear as beautiful objects; these villous tufts are

of a light pink, looking very much like anemones when seen in the illuminated tanks of an aquarium These papillomata arise from the vesical mucous membrane, and there may be one or many tufts in the bladder. When they are examined microscopically they have a very characteristic structure. They consist of an axis of delicate connective tissue containing blood-vessels, and over this, on a basement membrane, you will see a cap of epithelial cells of the same character as those which line the interior of the bladder. The villous tufts are very vascular, and if from any cause these cells are detached, these blood-vessels, which form the axis of the villous tuft, begin to bleed, and sometimes, if a tuft is near the urethra, it may be long enough to be carried into the urethra, block it up and cause great pain—almost as much pain as if a stone were impacted in the urethra. Papillomata are particularly apt to grow near the vesical orifices of the ureters. The unpleasant thing with regard to these villous papillomata is the fact that they very often become malignant, and sometimes in that particular portion of the bladder from which the villous disease grows the cells take on malignant action and penetrate the bladder wall. And, indeed, some surgeons, who devote their attention to the bladder, prefer, unless these papillomata are causing great trouble, to let them alone, because they are of opinion that by detaching them you injure the integrity of the mucous membrane of the bladder, and conditions may be set up which become malignant.

My own experience teaches me to regard a villous papilloma as a pre-cancerous condition, but as in other conditions of this kind, they may exist for many years and never become cancerous. Many years ago, when I was a dresser, I remember quite well an old man who had been at intervals in the hospital over a period of seventeen years on account of hæmaturia, which was regarded—and correctly—as being due to villous disease, because when he came in with these attacks we used to find fragments of the villi in his urine, so large that they did not require any magnifying glass to see them. About that time (1880) Sir Henry Thompson advocated perinæal section and removal of these villous tumours, and this old man was subjected to that operation. He was old, and he died from the operation. This is his bladder, and it contains a large tuft of villous papilloma. Another very remarkable thing about these cells on the tufts of the villi is their vitality. Those of you who are interested in the cytology of malignant growths cannot do better than examine the epithelial cells that lie on the top of these villi. They show all those remarkable changes which are known as the mitotic changes in a very remarkable manner, and they also illustrate in a very striking way the power which these cells have of engrafting themselves on to the tissue with which they come in contact. You know that the pelvis of the kidney is lined with epithelium, and from this villous papillomata may grow like those arising from the mucous membrane of the bladder. Our museum contains two admirable examples. As in the case of the bladder, these villous tufts are often detached, and the fragments are conveyed down the ureter by the urine, and in their passage they cause pain and distress exactly like that produced by a calculus when it passes down the ureter. They are not common, but a fair number of cases are known, and kidneys have even been removed because of them. And some enterprising surgeons, when they have opened the kidney and found its pelvis full of villous tufts, have opened the pelvis of the kidney, removed the papillomata, and saved the kidney. Such efforts of conservative surgery have been rewarded with success. But these cases in the kidney are also interesting from another point of view. Here is a specimen which was described by Dr. Murchison, a very distinguished physician, who at one time was on the staff of this hospital. In making the post-mortem he discovered that the patient had got villous papillomata in the kidney, and some similar tufts were found on the vesical mucous membrane near the orifice of the ureter. Murchison believed that these were due to cells which had descended the ureter with the urine, and had engrafted themselves on the mucous membrane of the bladder. Now, to finish with the case of the old lady. We took her into the theatre, and we discovered that she had papillomata in her bladder, and with the assistance of Sim's speculum we opened the bladder through its posterior wall and removed the villous tufts. We knew from our first examination that there were two, but when the bladder was opened through the vagina we were surprised to find they were so small, and I was so anxious for fear I might miss some that we spent a long time moving our fingers about to discover

some more. After we had removed the tufts we sewed the bladder up, and my house-surgeon and myself were particularly anxious to see the condition of the urine next morning, and to our satisfaction we found the urine free from blood.

Now we pass from these papillomata with rich epithelium, which grow in the bladder and in the kidney, to another extremely interesting kind of villous papillomata, namely, those which grow into the interior of cysts in the immediate neighbourhood of the ovary. There is a certain kind of cyst which grows between the layers of the mesosalpinx; such cysts are extremely prone to have papillomata on their inner surface: these warts may occur here and there, or they may form definite clumps, which sometimes become calcified, or they may form huge dendritic masses inside the cyst until, merely by their power of growth, they burst the cyst open, and then you see the whole of that surface, which the cyst formerly occupied, replaced by a thing which looks like a cauliflower. These papillomata in the cysts are vascular, though perhaps not quite so vascular as the vesical papillomata, and when the cyst is ruptured in this way the movements of the intestines detach these cells, and they will be strewn over the pelvic peritoneum, and drop on the surface of the bowels and in the recesses of the peritoneum, on which they engraft themselves and grow into papillomata. Crops of peritoneal warts of this kind are always accompanied by a large amount of free fluid in the abdomen, which is known as hydroperitoneum. And if you let off the fluid and remove these papillomatous cysts the fluid does not reaccumulate. It has been proved over and over again that these warts, which lie about on the intestines, will then atrophy, like the warts on the hands of children. As long as the parent tumour is in the abdomen these cells continue to be detached, and, although detached warts may die away, fresh crops come in their place. I have mentioned this matter, too, in order to demonstrate the extreme vitality of epithelial cells. This leads me to consider that most remarkable and most vital of all cells known as the ovum or sex cell. The ovum may be practically regarded as an epithelial cell shed from the ovarian follicle. It is a large cell which is surrounded by a number of smaller cells in the ovarian follicle, and the ovum itself is derived from the cells which cover the embryonic ovary, and later become embedded in its stroma. The sex cell, or ovum, is an epithelial cell. We began this lecture by contemplating a bottle of dirty urine, and by tracing its causes we find ourselves face to face with the great problem of life, which centres around the ovum.

January 11th, 1909.

Prevention of Recurrence of Kidney Stones.—Klemperer expatiates on the necessity for internal medical measures to prevent recurrence of kidney stones after their operative removal, and advocates for this measures to promote the flow of urine through the kidneys to wash out the sediment that otherwise might form concretions. Every such patient should be advised, he asserts, to drink frequently between meals, taking from 200 to 300 c.c. every two or three hours and to drink copiously just before retiring, as the night urine is the most concentrated and has the greatest tendency to sediment formation. A glass of water beside the bed at night can be sipped as the patient wakes—such persons are generally elderly and wake often. The fate of these patients depends on the observance of this advice. The kind of beverage is of less importance, although weakly alkaline, carbonated mineral waters should be given the preference. He adds that from the standpoint of prevention of recurrence of kidney stones there are no contra-indications to the use of alcoholic beverages. The patients with a tendency to obesity and heart disease should drink copiously, as above, but exclusively between meals, taking food frequently, but in small amounts, that leave the stomach soon empty, and drinking freely between the meals. In this way the tendency to kidney stones and to obesity will be combated at the same time. Rosenfeld has his obese patients drink up to ten quarts of water a day, all between meals, and has found this extremely effectual in reducing weight. In heart disease, the free ingestion of water as above is not advisable in case of failing compensation, but in other conditions it is not contra-indicated. The intake of fluid within reasonable bounds does not injure the heart muscle, as is shown by the blood pressure and the experiences with diabetes. The diet should be mixed, but the oxalate-stone patients should refrain from tea and spinach, and the urate-stone patients from eating the glandular organs (thymus, liver, kidneys).—*Journ. A.M.A.*, vol. li, No. 25.

THE DIAGNOSIS AND TREATMENT OF SCIATICA.*

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SCIATIC neuritis, or sciatica, may be produced by a variety of causes, which, for the purposes of classification, may be described as (1) primary and (2) secondary. The latter group will include cases in which the nerve or the nerve-sheath has become involved by pressure or by an inflammatory process in the pelvis or buttock, such as a pelvic tumour, lumbo-sacral osteo-arthritis, tubercular caries of the sacrum or lower lumbar vertebræ, caries of the ilium, or chronic osteo-arthritis of the hip-joint. In chronic osteo-arthritis of the lumbar spine, sacrum, or hip-joint, a good X-ray photograph will very probably be able to confirm the diagnosis. In all of the above conditions the signs and symptoms of a definite sciatic neuritis may be present, and it will therefore be necessary to exclude them by a systematic examination of the pelvis, spine, and hip-joint before making our diagnosis of a primary sciatica. Further, in tabes dorsalis, chronic Bright's disease, and peripheral neuritis due to diabetes or to chronic alcoholism, bilateral pains, more or less along the course of the sciatic nerve, may be mistaken for a genuine primary sciatica. This bilateral feature of the pains is, indeed, the most suggestive indication that some other cause than a primary sciatica is to be looked for, such as tabes, peripheral neuritis, chronic osteo-arthritis, or tubercular caries of the lower lumbar vertebræ or sacrum, or possibly a spinal tumour or pachymeningitis. *Syphilis* is a not very uncommon cause of severe pains around the lower back, hip, and front of the thigh, due to a gummatous neuritis affecting the lumbar roots, and causing wasting and fibrillary tremors in the muscles on the front of the thigh, with some diminution of sensation on the front of the thigh and leg. This disease appears to have a special predilection for the upper lumbar roots, distinguishing it from sciatica. There should be no difficulty at all in differentiating between these two diseases, inasmuch as the course of the pains differs from that of sciatica in invading the front of the thigh, and also in the rapid course of the disease, leading to noticeable wasting of the thigh within a few weeks. I have met with it only in men, and it usually yields quickly to energetic anti-syphilitic treatment. Indeed, so impressed am I with the relative frequency of this condition that I always try anti-syphilitic treatment in any case of recent and increasing pain around the hip and front of the thigh.

Gouty neuritis may cause more or less symmetrical pains around the hips and thighs, both front and back. This is often spoken of by the patient, and sometimes by his doctor, as sciatica, though it runs a somewhat different course. A gentleman, æt. 68 years, now convalescent, complained of chronic sciatica, to which he said he had been subject for about five years. On examination it turned out that the pains were bilateral, sometimes worse in one thigh and leg, sometimes in the other. Flexion of the hip with the knee kept extended, instead of increasing his discomfort and causing pain at the back of the buttock, as is almost invariably the case in true sciatica, gave him actual relief. The hip-joints were perfectly normal, examination of the rectum revealed no evidence of pelvic tumour, and in spite of the duration of the symptoms at intervals during five years there was no alteration in the reflexes, no muscular wasting or anæsthesia. His pains were quite different in type to those of sciatica, sudden spasms coming on even when at rest in bed, so sharp as to cause his facial muscles to twitch, and reminding one of the pains in tabes. He had no signs, however, of this disease, and there was no albumen or sugar in his urine.

In another case of bilateral pains the cause turned out after about nine months to be *tubercular caries* of the sacrum. A man, æt. 42 years, first felt pains in the left thigh after driving several miles in very cold weather, and he was laid up for three weeks with apparently typical sciatica, the pain then leaving the left side and attacking the right thigh, in which it persisted in spite of aspirin and rest in bed. He was then put on a water bed and treated with cataphoresis with salicylate of soda to the buttock, passing a strong current of 25 m.a. along the leg. He made a very good recovery, being quite free from pain in less than a fortnight. He then went to a spa for a course of baths, but

* Delivered before the Medical Society of London.

the pains returned, and were again relieved by the cataphoresis treatment. He recovered practically for nine months, when he developed definite signs of bilateral psoas abscess, which was operated on. In this case, although the signs and symptoms were those of typical sciatica, yet the one fact that the pains had affected both sides was very suspicious from the first of spinal disease. The first definite sign that there was grave mischief was the development of incontinence of both sphincters, with the appearance of some resistance on deep pressure in the right loin, but after a month the sphincter trouble cleared up.

Primary sciatica may be (*a*) acute or (*b*) chronic. (*a*) *Acute sciatica* may be defined as a rheumatic, or in some cases a gouty, *perineuritis* of the sheath of the sciatic nerve, either at its exit from the pelvis at the sciatic notch, or starting a little farther down and affecting the nerve, perhaps, for several inches down as far as the middle of the thigh. Two modes of onset are especially definite and common: (1) in which the sciatica is preceded by a lumbago, or lumbo-sacral fibrositis; (2) in which the attack is closely preceded by exposure of the back and thigh to cold and wet, as by sitting in wet clothes, tobogganing, etc.

The nerve-sheath is stated to be swollen, thickened and reddened at this stage, with, perhaps, an inflammatory exudate, and the symptoms of severe lancinating pains, worse on every movement, and especially of flexion of the hip, with tenderness on pressure over the course of the nerve, can easily be understood by the involvement of the nerve-filaments supplying the sheath in the inflammatory tissue. The tenderness is usually most marked at the fold of the buttock, or three or four inches further down the thigh. Irritation of the nerve-bundles passing upwards from the leg through this inflamed area causes referred pains along the course of the nerve down to the ankle, with, perhaps, the appearance of tender spots at the ankle or knee. At this stage there is no muscular weakness or wasting, no anæsthesia, and the only alteration of the reflexes is an occasional increased excitability of the Achilles jerk on the side of the affected leg. Extension of the nerve by flexing the hip with the knee kept straight causes severe pain, and the gait is apt to be characteristically altered. The patient walks with the body slightly bent forwards and towards the affected side; the knee is kept slightly flexed, and the pelvis is swung forward with the affected lower limb, in order to minimise the amount of movement of the hip, the foot being turned inwards. In the worst cases there is pain in any and every position, though it is worse on movement. Pain is present even when lying flat in bed, and the patient cannot lie on the affected side, but usually prefers to lie slightly turned over towards the other side, the bad leg being supported by the other, or on a cushion placed between them. Sleep is difficult and broken, and may often best be relieved by suppositories of half a grain of morphia at 9 p.m. There are some points in the treatment which I consider especially useful at this stage: the patient should be kept entirely in bed upon a full-sized *water or air-mattress*, taking care to place a double layer of blanket between the rubber mattress and the under-sheet for the patient to lie on. This alone gives considerable relief, and may allow a patient to lie on his back who could previously lie only turned over on the sound side.

Galvanic treatment.—It is often taught that galvanism should not be used until the acute stage has passed, but in my opinion a most valuable remedy is then withheld. A strong current should be used, but it must be given with great care, and the effects are much better if cataphoresis with salicylate of soda is employed. My practice is to use a flat padded leaden electrode, seven inches by four, placed longitudinally across the fold of the buttock, the face of the electrode being covered with a layer of wet cotton-wool, over which is poured a couple of drachms of saturated solution of salicylate of soda. The electrode is attached by a length of electric-bell wire to the negative binding screw of a galvanic battery of 18 or more cells. The positive binding screw is similarly attached to a larger electrode, which is placed either at the back of the lower part of the thigh, or is moulded around the lower part of the leg and ankle, according as to which is the more painful spot. This lower electrode is similarly padded with wet cotton-wool, on which is placed some bicarbonate of soda to make a thin paste. Owing to the projection of the bones at the ankle-joint it is almost impossible to mould a leaden electrode so as to press evenly on the skin, and there is consequently considerable danger of electrolysis of the skin in

this region, hence a more flexible electrode made of copper ring-mail, thickly padded, I find more satisfactory to use for the ankle. With the electrodes placed in position, the leg resting on a thick towel placed over a mackintosh and warmly covered up, the current is turned on slowly until from 20 to 25 or even 30 milliampères are registered on the galvanometer. With a newly-charged battery, the electrodes being applied in this manner, this strength of current may be obtained from thirteen to fifteen cells. The current should be allowed to run for about twenty minutes, and the process should be repeated twice a day, morning and evening. The effect of using salicylate of soda upon the negative electrode is to force the electro-negative salicyl ions through the skin into the tissues on their way to the positive electrode; in this way a drug, if of the nature of an acid or base, may be made to enter the tissues from the appropriate electrode, negative or positive as the case may be, and exert a local and a general action upon the tissues. Aspirin may also be given with advantage by the mouth in 15-grain doses three times a day, and relief from the severity of the pain is to be looked for from the first day, improvement in easy lying and in length of sleep at night being the first signs of improvement. This treatment should be continued uninterruptedly for a fortnight, with the exception of the morphia suppositories, which may usually be discarded after the first four or five days. The object of the treatment is to cause the arrest of the perineuritis, the absorption of any inflammatory exudate, and lastly, but by no means least, to prevent the formation of adhesions between the sheath of the nerve and surrounding structures. The first two of these points will be best gained, in my opinion, by the treatment I have described above, but in order to prevent the formation of adhesions it is necessary to use *massage and passive movements* of the hip at the right time. If massage is used too early during the acute inflammatory stage of the sheath it is obvious that harm and not good will be done, whereas if it be omitted altogether, or postponed for several weeks, there is a danger that adhesions may form, too tough to be broken down except by a surgical operation. It is, therefore, a somewhat nice point to decide precisely when to commence the massage and movements. I usually do so at the end of the fortnight's treatment, when the water mattress may be removed, and the galvanism given only once daily. The indications for the commencement of the massage are the disappearance of all pain while lying quiet, and the recovery of ability to lie on the affected side, together with the marked diminution or disappearance of tenderness of the nerve on pressure, with an increased range of flexion of the hip, keeping the knee straight, without causing pain at the back of the buttock. At the same time the patient is now allowed to get up, at first for an hour a day only, to walk about the room, and to perform certain exercises, namely, to stand on the affected leg, swinging the other straight forwards; to bend down in the endeavour to touch the toes, keeping the knees straight. The after treatment is also important in prophylaxis against recurrence, namely, protection against chill by wearing chamois-leather pants outside the ordinary ones, and avoidance of sitting on cold seats, or chairs with hard, unpadded fronts, such as deck-chairs.

There are three other types of case to which I wish to refer:

(1) Those in whom an aching pain is present only upon lying down or sitting, while the patients are able to walk, dance, row, etc., without feeling any discomfort. These are the slighter cases of perineuritis, and they always develop gradually, not acutely. The absence of pain on movement is due to the consequent mental distraction. These cases do excellently well under the same treatment I have described for acute perineuritis, rest on a water-bed, with strong cataphoresis with salicylate of soda over the nerve, and subsequent massage and passive movements.

(2) *Sciatic neuralgia.*—In this much rarer type the pain is purely neuralgic, though it may be a sequel to an attack of acute sciatica. The following is an excellent instance of this form. A young soldier, after contracting typhoid fever in Africa, developed sciatica on his way home. He was treated with complete rest in bed for two months after his arrival, the pain being absent while lying down, though returning at once if he got up. This condition continued for eight months without any improvement, rest in bed in a hospital having been tried again in the interval. On examination I found no pain on stretching the nerve, though he had marked tenderness at various points along its

course. He came to the Out-patient Department of St. Mary's, where he had strong galvanism given him daily for a fortnight, remaining up and about in the meantime. By the tenth day his pain had completely disappeared, and had not recurred three weeks later.

Chronic sciatica.—By attention to the details of treatment described above I am convinced very few cases of acute sciatica would drag on into chronic cases with constant pain and wasting of the muscles, which denote that the original perineuritis has invaded the nerve-bundles, setting up an interstitial neuritis and damaging the actual nerve-fibres by pressure, as the inflammatory tissue hardens down into fibrous tissue. In this stage of chronic sciatica the whole nerve may become toughened and shrunken, and is then curable only by free longitudinal incision of the nerve-sheath, and splitting up the nerve into its constituent bundles, so as to free the nerve-fibres from the strangling effect of the fibrous formation which has resulted from the interstitial neuritis. Such a condition of the nerve Mr. Pepper informs me he found in the case of a gentleman who had been treated for years for chronic sciatica, whose pain was constant, and the tenderness on pressure over the nerve intense. His constant assumption of the flexed position of the body and hip to relieve his pain had produced organic contracture of the muscles and fasciæ, which did not disappear under an anæsthetic, but ultimately recovered under orthopædic treatment after the nerve had been freely incised from a point underneath the gluteus maximus down to the middle of the thigh.

Thus there are two conditions of the nerve which may be relieved surgically:

(1) External adhesions between the sheath and surrounding structures, from the sciatic notch down to the middle of the thigh.

(2) The toughened and contracted condition of the nerve itself, which results from those severe cases of perineuritis which have gone on to chronic interstitial neuritis.

Clinically, these two classes of cases of chronic sciatica will differ notably: (1) Those with external adhesions only will be perfectly free from pain on lying down or standing still, but may suffer acutely after walking. Moreover, there will be no wasting of the muscles, no anæsthesia, and no alteration of the Achilles jerk.

(2) In the rarer cases with a toughened and contracted nerve the pain will be more or less constant, though it is increased on movement. There will be marked tenderness of the nerve on pressure over various points along its course from the sciatic notch to the middle of the thigh, and there will be muscular wasting of the leg, with anæsthesia and loss of the Achilles jerk.

Treatment by nerve-stretching, acupuncture, and injections of all kinds are, I believe, valueless as they are unscientific for the treatment of a mixed motor and sensory nerve such as the sciatic. Blisters down the back of the thigh have certainly more to be said for them, though, personally, I prefer to do without them.

In conclusion, I strongly deplore the pessimistic attitude adopted by some towards the treatment of sciatica, and that patients should be told that they will have to bear the pain until the disease wears itself out indicates a cynical frame of mind which is not justified by modern knowledge of the pathology of sciatica, and of the treatments based upon it.

Dr. C. W. CHAPMAN said he had had two very severe attacks of sciatica, and therefore had taken a great interest in the subject. He had great faith in the early application of blisters, as soon as the pain began to be felt, and a calomel purge. That treatment would often cut short an attack. Dr. Harris did not mention coal-tar preparations in combatting the pain. He (Dr. Chapman) had never used morphia, and he had never found coal-tar derivatives fail. He felt very grateful for the suggestion concerning the water-bed, and also that concerning chamois-leather pants. Cataphoresis did very much good for the condition: he had had it done with iodine paint with marked benefit, especially when the pain attacked the fascia of the thigh. He had also taken iodide of potassium internally in very large doses, up to 15 grains thrice daily.

Sir JOHN BROADBENT asked whether Dr. Harris had any experience of ice-bags in the condition. He had known the treatment give very considerable relief in the condition, though patients generally objected because they said it was cold which was the cause of the condition.

Dr. VINCENT BELL asked whether Dr. Harris had any experience of the subcutaneous injection of strychnine for checking the wasting of the muscles in the condition.

Mr. C. B. LOCKWOOD said a little while ago he had a curious adventure with a case of sciatica. A woman had had the rectum removed for malignant disease. A year afterwards she suffered very great pain in the left sciatic nerve. This was exposed to the great sciatic notch and a carcinoma felt inside her pelvis in the proximity to the nerve, which was freed. That proceeding relieved her pain for a time, but afterwards it became so agonising that it was considered justifiable to take extreme measures, and he opened her spinal theca and removed the posterior roots of the spinal nerves which went to the great sciatic. That operation entirely removed the pain throughout the distribution of the great sciatic nerve. But very soon she suffered intolerable pain in the joints of her left limb, and on inquiry he found that it had been said that the sensations from the joints did not pass up the posterior roots of the nerve like ordinary sensations, but up the anterior. Therefore he did not think it advisable on another occasion to remove the posterior roots of the nerve to stop pain of that description.

Dr. HARRIS, in reply, said that as a rule he tried to do without coal-tar preparations in the acute stage, because that seemed to be merely driving away the pain for the time and obscuring the symptoms. He did not use morphia, except for procuring sleep at night, and then he gave it by the bowel. He had been very interested in what was said about ice-bags. He had not used them for sciatica, and he would hesitate to do so, because one heard such very definite stories of exposure to cold producing the sciatica. He could only explain the marked relief which Sir John Broadbent referred to as following on ice-bags by supposing that the continuous application of cold was different from the exposure to a cold wind. On examining the skin under an ice-bag, after it had been there half an hour or so, it would be seen to present the same appearance as after a poultice, and probably the relief of pain was due to the same cause, namely, the flushing of the skin area by extra blood. Strychnine he had used for the improvement of muscular wasting, only in conjunction with treatment to the nerve. He did not think strychnine alone would stop muscular wasting. He had been much interested in the President's remark about division of the posterior roots. He (Dr. Harris) had not heard of that intense pain produced in joints by posterior nerve-root division, although he had known cases in which it had been done.

Sherrington's work on arthritic muscular atrophy certainly proves that the posterior spinal roots carry afferent sensory impressions from the joints.

January 11th, 1909.

Loss of Appetite in Children.—I. M. Snow calls attention to a group of symptoms observed in children from two to ten years of age, characterised by capricious and lacking appetite in spite of the most correct regimen and attractive food, irritability, lack of nerve, irregular and unfreshing sleep, and general deterioration in physique. All this may be the case in spite of tonic, fresh air treatment, and sometimes rapid growth without corresponding increase in weight. The condition is to be distinguished from the anorexia nervosa of Gull, which is a type of hysteria with loss of weight without organic lesion and with delusions and marked hysterical manifestations, which are lacking in these cases. Snow insists on a thorough examination in these cases excluding all organic diseases, and finds the efficient treatment only in putting the child to bed and keeping him there for two weeks with plenty of ventilation and fresh air. The body should be sponged off and well rubbed morning and evening and a suitable nutritious diet, well cooked and attractive, may be given without any special restrictions. The rest treatment should be continued until there is considerable gain in weight, a good appetite and natural sleep for ten or twelve days. These being satisfactory, the child is dressed and given an hour's perfect freedom, out of doors if preferred, and if improvement continues this may be increased by an hour each day until the child has six hours in the middle of the day.—*Medical Record*, vol. lxxiv, No. 25.

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A LECTURE

ON

MALARIA.

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MALARIA, unlike some other diseases, helps you at the start by its very name, derived from two Italian words—*mal aria* (bad air). There was an old idea, which still prevails in some parts of Italy and Spain that if you opened your window at night you let foul air in, and with this air came something that they christened miasma or malaria. Many people seem to have this idea still, and it may be one of the reasons, in those countries where malaria used to be rife, why people are somewhat afraid of opening their windows and therefore sleep with them closed. Our ancestors, who gave the name to this disease, were searchers after truth, but they had not quite reached it. It is quite true that something came in at night and poisoned them and gave them fever, but it was not the air but the gnats or mosquitoes, that loved the darkness of night and not the light of day.

I will take next the French word for this disease, *paludisme*, derived from the Latin, the English equivalents being swamp or marsh fever. In either case the idea is the same—the horrible miasma which people thought they could see. In such books as 'Westward Ho,' for example, people in the ship on the sea looking at the land saw the malarial miasma. Whenever sailors were sent on shore in the tropics they caught this disease.

The English name (ague) is not a bad one, because it does not commit you to any pathological proposition. It is taken from the French word *aigu*, which means sharp. Anyone who has seen an attack knows that it is really a sudden and acute manifestation of intermittent fever where the temperature goes up and down at intervals, or we call it

remittent fever when the fever abates but does not cease. We call it also tertian and quartan. Tertian means that you skip one day, that you have the disease, for instance, on Monday and Wednesday and Friday and you skip Tuesday and Thursday. It is called tertian because it occurs again on the third day. Then quartan means that the fever skipped the second and third day and came on the fourth. We have a third disease, for besides the ordinary benign or simple tertian and quartan there is the malignant form of the disease, which the Italians call the summer-autumnal (æstivo-autumnal). The English call it malignant tertian or sub-tertian.

There are two or three terms which the public have invented. Climatic fever is one. They will say that they suffer from the fever of the place in which they live. If they are living, for instance, in Jamaica or Bermuda, they tell you they have Jamaica or Bermuda fever, or they say they have "plain fever." There is the "hill fever," which means that certain mosquitoes have followed their human hosts into the hills. Then, again, there is "jungle fever," "river fever," and there is a modern American term, "gnat fever." That is the best name of all.

Definition.—A specific infectious disease, produced by certain protozoal parasites, whose host is the mosquito, but whose intermediate host is man. These parasites inhabit the red blood-corpuscles of man and give rise to fever, anæmia, enlargement of the spleen, and deposit a black pigment in the internal organs and elsewhere. The main clinical signs you have to recognise are fever, anæmia, and enlargement of the spleen.

I will not weary you with the history of malaria, because it is an old story, and goes back to the days of Hippocrates, 400 B.C., and Celsus, who lived in the first century of the Christian era. To-day everybody knows that quinine is given for malaria, and that quinine is an alkaloid from the cinchona bark. But malarial fevers were never scientifically studied until about 1639, when the cinchona bark was imported into Europe by the Countess of Chinchon, the wife of the Viceroy of Peru, who had been cured of malaria by it. The bark was introduced into England and cured Charles II of tertian fever, as well as the Dauphin of France; and after that people began to be more interested in malaria and its cure by quinine. It was about 1712 that the term "malaria" was first used. In 1880, coming on to more modern times, Laveran, in Algeria, discovered the parasite in the malarial blood, and he put both the diagnosis and treatment of malaria on a scientific basis. All honour to him, because he was working with very poor microscopes, and yet he was able to find what other people were looking for in vain. In 1897 Major Ronald Ross proved that the *anopheles* mosquito was the infecting agent. In 1900 it was thought by the London School of Tropical Medicine that it should be definitely proved to the public that the mosquito did convey malaria, so some of the staff of the hospital went to live in the Campagna in Italy in houses protected by wire gauze from mosquitoes. They lived there perfectly happily and comfortably, and without taking quinine, and did not contract malaria, although the peasants all around were suffering from the disease. Another experiment succeeded in producing malaria by introducing infected mosquitoes into London. By special arrangement with the Italian Government some mosquitoes, which had been fed on malarial patients, were sent by post, and they were allowed to feed here on men who had never suffered from malaria, and had never been out of England. These volunteers suffered from malaria, and one of them is still at the Tropical School, aud proudly shows lantern slides taken from his own blood when he caught malaria. I will pass round a map of the geographical distribution of malaria, from which you will see, roughly speaking, where malarial disease lurks—in hot or warmish countries. I should say that the tertian fever is quite a common one, the quartan is rare, and we will not talk much about it to-day, while the benign tertian and the pernicious or malignant tertian are both common. It is quite possible for fever to occur every day, when it is called quotidian, because you have two separate broods, as it were: you have not only a brood which attacks you on Monday, Wednesday, and Friday, but another which attacks you on Tuesday, Thursday, and Saturday. So that every day there comes an attack of fever.

Long before we knew that there were such things as malarial parasites we knew that quinine was good for them, but we did not know why quinine was useful and why men suffering from malaria became anæmic. We now know that the

parasite gets inside the red corpuscles and eats the hæmoglobin. That is why persons, returning from the tropics, have generally a whitish or sallow appearance compared with the red countenance of healthy people in this country. We also know that quinine kills the malarial parasite.

There are two phases in the existence of the malarial parasite. There is the one inside man—the human cycle; there is the other inside the mosquito—the mosquito cycle. If you examine the blood of a malarial patient one or two hours before the rigor, you will find that the parasite is a pale disc of protoplasm occupying a large or small area within some of the red blood-corpuscles, and scattered through this pale disc are a number of particles of blackish pigment.

You will wish me to say a few words about the mosquitoes. They belong to the order of what are called diptera, or two-winged insects, or true flies. They differ from all other flies because they have scales on their wings and body, and in all the kinds which concern us there is a long piercing proboscis. Mosquitoes are divided into several different families. The two which are usually chosen by way of contrast are the *culex*, which is harmless as regards malaria, and the *anopheles*, which is dangerous. Here is a picture of a full-grown insect, and you will observe that it has a hunch-back appearance. The *culex* looks as if it were clinging to the wall with all its legs, whereas this other is rather standing on its head in an irreverent fashion. There is a difference in the antennæ. The male insect has a more beautiful plumage than the female in both cases. There is a difference in the eggs and also in the larvæ. Here in this picture is an individual killing mosquitoes in a stagnant marsh, and here are other people living in a house well screened from mosquitoes. I am not going to bother you with the names of the various mosquitoes that will give you malaria. There are a certain number of them, and they usually belong to the family of the *anopheles*. The kinds of mosquito vary in different parts of the tropical world. I may remind you that it is only the female that bites; the male is a harmless, inoffensive insect, and feeds on bananas and other fruit juices. The normal diet of the female is blood—human blood or cattle blood every night when she can get it. Some of the females bite during the day, but their feeding time is usually after dusk and before dawn, somewhere about 3 o'clock in the morning. They prefer certain dark colours and something that smells; thus they like a brown or black tint and nothing very light. With regard to smells, they like old boots and blacking and are fond of negro skins.

As to their life, I daresay you have read that their existence is ephemeral, or perhaps that the mosquito lives for years. Neither of those statements is quite true. It has been found to live one or two months in the dark corners of native huts. It will live many weeks in captivity if properly housed and fed on bananas. Mosquitoes love heat, and therefore people suffer from malaria all the year round in tropical places and during summer and autumn in temperate countries, because mosquitoes are then more common. Just now in India there is a serious epidemic of malaria in the Punjab, where, for the last few months, since the cessation of heavy monsoon rains, the epidemic has become so terrible that in some places the people are dying at the rate of 100 a day, and at Lahore 60 drivers and 2000 mechanics are on the sick-list of the railway staff. Mosquitoes prefer low levels and dislike breezes, therefore the inhabitants of lower stories are more infected than those living in upper stories. There are, however, certain mosquitoes which prefer hilly situations. If you want to get safely away from mosquitoes in tropical countries you must go to a height of at least 5000 feet above the sea-level. Moisture I have touched upon already. Marshes and low-lying ground are conducive to malaria, because the mosquitoes are more numerous there and breed best in moist places. Continuous moderate rain favours malaria, because it lessens the resistance of man and favours the breeding of the mosquito; but tremendous tropical rain kills mosquitoes. It is generally after the rain that mosquitoes flourish, when they come out and live in puddles which have been left by the rain. The hoof-marks of cattle going to water over soft ground make a delightful home for the female mosquito to lay her eggs in, where she is certain that her offspring will not be drowned or overcome by strong winds. In the same way they get into wet tins, or bottles, or anything else left in back-yards of houses.

The old idea was that malarial miasma came from the soil. Any old Indian, if questioned, will tell you that. When the ground is turned up in

certain places to make railways and the men all suffer from malaria, nothing will persuade him that the disease did not come out of the ground. But, as a matter of fact, when damp ground is turned up there must be a certain amount of moisture; even if there is not rain or the inundation of a river, there are pools of water in which mosquitoes breed. The origin of that Indian idea was that sailors cruising in tropical seas were found not to contract malaria till they landed, or until they came within the mosquito zone. The mosquito does not fly out more than a quarter of a mile to meet a ship. It was, therefore, thought that when these men landed they found the upturned soil and the tropical low marshy land, covered with pools of stagnant water, and that these factors caused malarial infection. But the soil, as soil, has nothing to do with malaria any more than the water has. It all depends upon what is in the water. Probably you all remember de Lesseps' attempt to make the Panama Canal. He had to give it up, because of the malaria and dysentery and yellow fever which destroyed his workers. Thousands of men literally laid down their lives in trying to make the Panama Canal. But to-day the Panama Canal is being pushed forward by American energy and knowledge, and the health of the workmen is as satisfactory as it is in New York. The difference is that they have learnt to kill the mosquito, and they are killing it as rapidly as they can and very thoroughly. We can put it plainly by saying that it is not the heat of the tropics or of warm countries which interferes with us humans; it is the animal life, the insects and worms of those countries, which give us diseases. We blame the country—it would be more accurate if we blamed the fauna of the country and not the air or the climate itself.

One of the myths—I have touched on a few—which you have heard is that black races are immune to malaria. This is absolutely untrue, but one or two facts have to be remembered. Black children are exposed to malaria more than the white children, because the white man, as a rule, does not go out to these countries as a child, but waits till he is a full-grown man. Then he compares himself with the black man of his own age. But the black man has already suffered from malaria, and, though some of his brothers and sisters have died of it as babes, he has survived and obtained a certain amount of immunity which makes him, of course, self-resistant to malaria. Children, as a matter of fact, are more susceptible to this disease than grown-up people, whatever the colour is, for mosquitoes prefer them. People will tell you that they caught malaria because they drank dirty water from a well. But there is no evidence of malaria being conveyed to the human by drinking water, although the water may contain the adult mosquito or the larva.

Immunity from malaria can be acquired. If you meet certain robust people living in tropical countries they may tell you that they do not suffer from malaria. Long residence in a malarial country may, in a strong person who survives, confer on him a relative immunity from the disease. They become salted, so to speak, by repeated attacks. This immunity is destroyed by ill-health, by hardship, and very often by removal to a fresh malarial locality. A person may become salted to his own kind of mosquito, but he is not necessarily salted to a species of mosquito occurring in another place. Least of all is he salted to black-water fever, about which I have not time to talk to-day. On the contrary, he is the man, above all others, who is liable to suffer from black-water fever. The man who gets black-water fever is nearly always one who has been living two or more years in a tropical place after many attacks of neglected malaria. He becomes filled with malarial germs and gets black-water fever, because I think black-water fever is only a complication of malaria, and not a disease by itself.

In talking of symptoms, you know what we mean by incubation—the time between the mosquito puncture and the first symptom of fever. That may vary from nine to twelve days. But a fresh comer into a malarial country generally waits one or two months before he is attacked I do not think it is due to any politeness or courtesy on the part of the lady mosquito. But 95 per cent. of the American soldiers who landed in Cuba gave a history of being there for periods varying from two to six weeks before they got their first rigor; the average time was one month.

All malarial fevers are intermittent in character, only becoming remittent after more than one attack. The tertian malaria is the commonest and mildest form in both tropical and temperate climates. If you talk to people who have come home from the tropics you may find that they have

very diverse views about the importance of malaria. You should remember always that you may be talking to people who have never seen any cases except those of benign tertian, which never kills. On the other hand, you may meet with persons who have returned from the West Coast of Africa, and have met with very few cases of malaria, excepting malignant tertian, which kills. That is why some people are more careful than others, and why mosquito nets and houses with other forms of protection are increasing on the West Coast of Africa, and are often neglected, I am ashamed to say, in our own British India, because Anglo-Indians do not understand the grave importance of the disease.

Those of you who have not yet had an opportunity of suffering from malaria have to be reminded that the paroxysm of fever occurs every forty-eight hours, and the onset of the paroxysm, that is, the rigor, is due to, and occurs during, the segmentation period or the splitting up of the parasite. The paroxysms consist of four stages—cold, heat, sweating and debility. The premonitory symptoms include malaise, yawning, nausea and headache, when the patient says, "I think I am going to have an attack of fever." Shortly after I began to study malaria, a girl from Demerara came to me, and on inquiring as to her first symptom, she said, "I feel so small that I could creep into anything!" After a few hours' discomfort there is a severe rigor that shakes the bed and makes the teeth chatter. In mild cases there is no rigor, but the patient complains of a chilly sensation as if water were running down the back. The rigor is most likely to occur between 12 noon and 8 p.m., and the temperature varies between 103° and 106° F., the skin feels like goose-skin, the lips and nails are blue, the pulse is rapid, there is headache, and the spleen increases in size. This stage lasts from a quarter of an hour to two hours. In children you may have convulsions instead of a rigor.

Then you come to the hot stage. During the cold stage you cannot get near enough to the fire and you cannot get enough blankets to put on you in bed; you can never be happy, even with hot-water bottles. In the hot stage you get a pleasant glow and begin to throw off your blankets, and you are perfectly happy until you get to the burning fever stage, and then you become uncomfortable again with a severe throbbing in the head, your skin is red and your conjunctiva red also, the pulse is full and bounding, the respirations rapid, and perhaps you have a dry cough. In severe cases you get delirium. This second stage usually lasts from four to six hours.

Next you get the sweating stage. As the fever subsides a heavy perspiration breaks out all over the body and the bed clothing is soon saturated. This lasts two or three hours, and at the end of the time the temperature is subnormal and the patient sleeps, the pulse is slow and weak, and in rare cases there may be a collapse. Those are the three stages in the attack. I always add a fourth, and that is a condition of debility. You are perfectly unfit for work for the next twenty-four hours. Therefore, people who have had an attack of malaria should be put off duty for twenty-four hours in order to convalesce.

Quartan fever is rare, and of it I have no time to speak on this occasion.

Malignant malaria represents something like 75 per cent. of the cases you may meet with in some parts of the world. The temperature is often remittent and irregular, each paroxysm lasting about thirty-six hours and occurring every forty-eight hours. The stages are like those of benign tertian, except that pain and vomiting are more likely to be present and that sweating is less marked. The ordinary morning and evening temperature chart is misleading; observations taken every four hours show the occurrence of rigors which otherwise would be missed.

Many different conditions may occur as sequelæ of malaria. One I will mention: I call it functional malaria. A man who has been living in the tropics comes to you and he only knows one disease, which is malaria. When anything is the matter with him he is certain that it is his old enemy. A man who has once had malaria thinks everything is malaria—he sees all new diseases through malarial spectacles.

Typho-malaria is still talked about in books. That is an unscientific and inaccurate term. You may have typhoid or malaria or you may have both at the same time, but there is no such disease as typho-malaria or a special mixture of the two. That is no more possible to occur than a like mixture of measles and smallpox, although a person may have measles and smallpox at the same time and have both rashes out together.

The diagnosis of malaria by clinical symptoms alone is often difficult, because there may be a double infection. The examination of the blood is the all-important thing. One examination is not enough; if it is negative, you have to examine again at short intervals. It is quite easy to take blood-films, only it is not much use taking them when the patient has already dosed himself with quinine. Of course, when you are in doubt in some strange place far away from slides and microscopes, you can always determine for yourselves whether the patient is suffering from malaria or not by giving him quinine. If he has malaria, and the quinine is given in the proper way, it is quite certain that the patient's fever will yield. If he has a disease like typhoid or Mediterranean fever the temperature will go on; it may bow politely to your quinine but it certainly will not disappear. Of course in hospital work we diagnose doubtful typhoid by the Widal test. Liver abscess is very often confused with malaria. The liver is increased in size and tender, and there is very often a history of dysentery. The spleen is not necessarily large in a case of abscess of the liver, but it is large in a case of malaria.

The prognosis is quite good in malaria, both in tertian and quartan. It is only doubtful in the malignant or pernicious form, for the first two days. The temperature may be above 106° F., and that, of course, is dangerous, as it would be in any disease. At the Albert Dock Hospital, in connection with the London School of Tropical Medicine, there have been four deaths from malaria during the last eight years, representing 1 per cent. of the cases.

As to the treatment of malaria, quinine is the specific which is certain to cure. A few points in the treatment ought specially to be remembered. Rest is important, quinine is more effective when the patient is confined to bed, recovery is more certain, and the danger of pernicious symptoms is lessened. Rest in bed must always be insisted upon in cases of malignant forms of the disease.

Here are several temperature charts which show you that soon after you begin to give quinine the temperature will drop. Malaria is the easiest disease to cure, though not easy to prevent.

As regards diet, nourishing liquids should be given during the fever, and for twenty-four hours after the temperature becomes normal. Calomel in small doses should be taken either before the quinine or with it, until the bowels move freely. Calomel favours the action of the quinine. The room should be quiet and well ventilated, and the patient, for the sake of others, should be protected from mosquitoes, because, as he has malaria and mosquitoes are possibly flying round him, any *Anopheles* mosquito coming near him and afterwards going to a healthy person can convey the disease. Quinine acts most effectively on the malarial parasite from the time of the segmentation of the parasites until the pigmentation begins within the red blood-corpuscles.

There is something to say about the kind of quinine you should give. There are twenty different salts of quinine. I will mention five of them. First of all, sulphate, which is our oldest friend. You have probably often drunk it or poured it down the throats of patients. This salt of quinine is most commonly used because it is the cheapest and best known. The objections to it are, that it is difficult to digest and requires 800 parts of water to dissolve it, so that coated tablets have been known to pass through the patient undissolved. You are therefore obliged to dilute it with an acid—sulphuric acid—which is not a pleasant thing for the stomach of a sick man. But there is no reason why sulphate of quinine should be given, and there is every reason for not giving it. Hydrochloride, 1 part of which is soluble in 40 of water, is much better. Bi-hydrochloride is better still, because it is soluble—1 in 2 of water, but it is a little more expensive. The bi-sulphate is exactly the same price as the sulphate, and is soluble in 1 in 11 of water. This is now used in the British Navy and Army, and ought to be used in the hospitals of this country. Euquinine, which causes cinchonism (deafness and buzzing in the ears), is sometimes useful for children and delicate people. It is much more expensive than the other kinds of quinine.

The time to give quinine is important. When the paroxysm has begun or when a patient is in the middle of an attack, quinine will not check it and will only make him more uncomfortable. You should wait till the sweating stage has begun or until a period without fever before the next attack. If you do this, the quinine comes into contact with the youngest forms of parasites and acts most vigorously. Continue the quinine for at least a

month after all symptoms have ceased. If you do these things you will hear nothing from your patient about constantly recurring attacks of malaria and that old stupid myth that once having had malaria you must always have it.

There are several different ways of giving quinine. By the mouth is the most common method, either in solution, capsule, cachet, pill or powder. If you give the bi-hydrochlorhide you may use an uncoated tablet or tabloid, and it will be dissolved before leaving the mouth unless quickly swallowed. To give quinine *per rectum* is unsatisfactory, whether by enema or otherwise, because absorption is slow and some irritability is caused. Moreover, you have to give 50 per cent. more than by the mouth. If you cannot give it by the mouth you should give it by the intra-muscular method, not the hypodermic, which is apt to produce abscesses. Make it intra-muscular, injecting aseptically into the buttock, flank, chest, or somewhere else. Then the quinine will act very quickly and you will get no abscess, though you may get some local tenderness and hardness for a few weeks. If a patient is comatose and dying the intra-muscular method is not quick enough and doctors in the tropics employ the intra-venous method.

With regard to the adult dose of quinine, less than 5 gr. are in my view useless for malaria, and a waste of time. I should say that more than 30 gr. in twenty-four hours are also useless. Ten grains every two hours, that is, 30 gr. per day for two days running, if given after paroxysms, will cut short quartan and benign tertian varieties. As to the summer-autumnal fever, you should give 5 gr. every four hours for one day and then 5 gr. every six hours for two days more. If you give bi-hydrochloride by the intra-muscular method you may give 8 gr. every night for three nights and after the initial treatment repeat the quinine twice a week.

The patient's idiosyncrasy has to be considered. Some will tell you they cannot take quinine. Most of those are not telling the truth, and if they are telling the truth it is no use their going to a tropical country. I met a lady once who asked me for a tonic. I gave her 2 gr. of quinine three times a day. After two days I was asked to visit her and found her in bed ill, with blood in her mouth and on the pillow and with odd purpuric spots about her. I said to her, "Have you had this before?" "Yes, once, and curiously enough it was after a tonic, too." "What tonic was it last time." "I have the prescription in my desk." I found it was quinine. Some doctor in London had given her that, and this lady suffered twice from hæmorrhage simply because she had taken two or three two-grain doses of quinine. People who suffer from singing in the ears, deafness and headache can be helped to a considerable extent by taking dilute hydrobromic acid, 20 ♏ at a time, with each dose of quinine. The symptomatic treatment of malaria is warm drinks and extra clothing in the cold stage, and sponging in the hot stage. In the burning stage antipyretics mostly do harm by weakening the patient.

There is one thing more to say. Any old Indian will tell you that if he can only sweat he will be on the high road to recovery. He believes that the sweating is the way in which the poison leaves his body, therefore, he will pile on blankets and do everything he can to make himself sweat. Profuse perspiration, without quinine, will not cure him.

There is no time to go into the question of prophylaxis or prevention of the disease. That is either personal or domestic, or, as one might say, municipal, for the State. The personal methods concern all of you, and so do the domestic requirements. The first lesson of all is that the mosquito will puncture you usually at night, though possibly in the day-time. Therefore, avoid mosquitoes. If you use a net it must be carefully tucked in, it must have no holes in it, there must be no question of the possibility of mosquitoes getting in, or biting you through the net.

There is one other way in which the disease can be avoided—by using what is called in China a Swatow lamp, a lamp cleverly arranged in which you can kill mosquitoes. In Puritan days it used to be thought that one of the pleasures that Protestants would have in the next world would be from a happy cool retreat to look down upon the wicked frizzling below. I never thoroughly understood that delight until I got this lamp made in Egypt and succeeded in catching hundreds of mosquitoes. The pleasure of hearing them enter the flame with a delightful pop is a joy which I commend to you. Of course you must avoid mosquitoes after sundown, for they get up after sunset before you go to bed, and therefore you

have to protect yourselves between sunset and bed-time as well as when in bed.

As to the question of prophylactic quinine, the two best ways are, either to take five grains every night when you go to bed or ten grains on two successive nights a week, say ten grains on Monday and ten on Tuesday, and no more till next Monday and Tuesday. That may make you uncomfortable for two nights. But do not imagine that that precaution is anything like so good as a mosquito net and protecting the house by wire gauze. You have to protect the doors, skylight, windows, chimney, and all other places where the mosquito might possibly find an entrance.

January 18th, 1909.

Dysmenorrhœa.—Veit says that the condition of the endometrium is dependent upon ovulation, and many appearances that previously were looked upon as pathological are simply the physiological conditions present in the menstrual or post-menstrual time. This recent view diminishes the number of anatomical lesions which can be held responsible for the symptom dysmenorrhœa. Veit defines menstruation as the abortion of an unfertilised ovum; ovulation should naturally be followed by impregnation, but as it does not every time in the human race, the various phenomena of ovulation must be conquered, which adaptation is outwardly shown by the function of menstruation. A nervous individual may react abnormally to the impulses of ovulation; in her the phenomena of conquering such impulses may not occur, and the result is one of the forms of nervous dysmenorrhœa. Another form is due to the faulty development of the uterus with co-existent abundant sexual excitement; the latter leads to changes in the ovaries and in the endometrium, and to the symptom of dysmenorrhœa, which in this case depends upon both anatomical and functional disturbances. The third form is the purely mechanical one due to some local disease or anomaly of the uterus or of the neighbouring organs. The nervous forms of dysmenorrhœa are to be treated by measures affecting the general health or by directing the treatment upon some possible source of peripheral irritation, attention to which may have a salutary psychic effect.—*Med. Record*, vol. lxxv, No. 1.

SORE EYELIDS.

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If one may judge fairly from the advertisements that appear within the covers of the popular magazines of the day, it would seem that there is scarcely anything that is more highly valued than an agreeable personal appearance. And it is not a matter for wonder that this should be so; it is true we have proverbs that tell us "handsome is that handsome does," yet there is always an unconscious bias in the mind of even the most ethically disposed of moderns in favour of a pleasing exterior. A proper jewel should have a fair casket. Ancient literature abounds in quaint narratives telling how the fate of families and tribes turned on these matters of appearance; and I do not doubt that you are all familiar with the very human story of the courtship and matrimonial affairs of that primitive Hebrew chieftain named Jacob, and how the ancient writings suggest his disappointment at the manner in which the cunning Laban foisted upon him the elder and plainer of his two daughters by the note that "Leah was tender eyed." Leah had sore eyelids, and Jacob would rather have had the comelier Rachel.

I do not propose to give you a critical disquisition on the many and varied diseases that can produce sore eyelids in man—that would take the space of half a modern text-book on ophthalmology. What I propose is to take two kinds of sore eyelids which every doctor will be sure to see plenty of in any sort of practice—blepharitis and gouty conjunctivitis—and to compare and contrast these two conditions.

These two forms of eye disease are as great a foil or contrast as could well be found, for they are separate as the poles of our social conditions. The one is plentiful amongst the children of the poor and dirty, and the other is found most often amongst the elderly rich and over-fed.

Blepharitis.

Since there are more poor than rich it goes without saying that of the two diseases the disease

of the poor is the more common; it is in fact the commonest eye disease amongst children. Let any man interested in eye work make a round of the children in a large elementary school in any one of our great cities, and he will easily be convinced of the prevalence of blepharitis, and he will not be long in noting that the disease is frequent in proportion to the dirtiness of the children he examines.

Let me give you a few figures: In one district of London I examined 22,000 children in the elementary schools; amongst these children I found 351 cases of eye disease, not including errors of refraction. Of these 351 cases, 270, or nearly two thirds, were cases of blepharitis.

Again, in examining these numbers of school-children I was careful to classify the schools into groups, distinguishing those schools where the scholars were of average cleanliness from those whose scholars were below or above this average of cleanliness. I found that whilst there was a bare 1 per cent. of eye disease in schools with children of superior cleanliness, there was 3 per cent. of disease amongst those below the average of cleanliness, that is to say, that neglect of the care of the person multiplies three-fold the risk of disfiguring eye disease.

Ætiology.

To understand the disease the anatomy of the eyelid and its margin must be remembered. The lid is a fold of skin; it has all the accessory structures normal to the skin, but since the lids are bound to the constant service of the eye, the accessory structures are modified in a manner which shows how beautifully Nature adapts a part to a particular service.

We need consider three structures only: the hair, the sweat-glands, and the grease-glands. The hairs are relegated to the edges of the lids, where they act as a veritable *chevaux-de-frise*; the grease-glands are ranged in rows along the lid margins as the giant Meibomian glands, whilst the sweat-glands are tucked back into the upper conjunctival fold, where they appear as the tear-glands. The benefit of this arrangement is best understood when we see the discomforts of the loss of any of these structures. A lashless lid is a poor protection against the numerous foreign bodies scattered by the wind or the myriad flying insects of a summer evening, whilst an excess of hair, as we see in the happily rare cases of distichiasis—double row of lashes—is as bad if not worse.

The tear-glands and the giant grease-glands are complementary to each other. The tears continually wash the conjunctiva, and were it not for the effective manner in which the grease-glands waterproof the lid margins the tears would be continually pouring over on to the skin of the face, making the skin sodden and eczematous—a state of affairs which would not conduce to the health of the conjunctiva. Again, the eyelids are in constant motion; and the motion is not continuous like the rotation of an axle-bearing, it is jerky, the lid margins tap against each other at each blink. The great grease-glands, by the spread of their unctious contents, protect the lid margins from actual contact, and, therefore, from irritation. Think what would be the soreness of the lids if they were not so protected, for a very simple calculation will show that the lids come in contact some 5000 to 20,000 times in a single waking day of fifteen hours!

Under all ordinary conditions the eyelids perform their duties without distress, but when the conditions are adverse the margins become inflamed, just as an ill-adjusted engine-bearing gets heated. Three conditions are adverse: (1) Excessive dirt; (2) excessive blinking; (3) inflammations of the contiguous structures, skin or conjunctiva. Although I put these three conditions separately, sometimes all three conditions are combined; sometimes one alone is enough, or nearly enough, to set up a blepharitis.

Every surgeon knows that the skin of the cleanest person harbours micro-organisms—the *Staphylococcus albus* for the most part—and that those portions of the skin which are most hairy harbour the most organisms. The lid margins fall into this latter caregory; and besides, they are particularly liable to contamination with extraneous matter from the fingering and rubbing to which they are subjected.

Bacteriological cultivations I have made from the lid margins of clean children produced large numbers of colonies of five different sorts of organisms, and similar cultivations from the lid margins in dirty children showed larger numbers of the same organisms, and particularly of those pyogenic types which are most often connected with pustular inflammations.

But excessive dirt alone is rarely enough to start a blepharitis; something besides is wanted to start the inflammation. And for this there is nothing like an acute specific fever, such as measles, or else of something quite different—the long-continued irritation of defective vision consequent on an error of refraction.

In an attack of conjunctivitis, such as is an almost constant effect of measles, there is a coincident change in the activity of the Meibomian glands. There is a distinct over-action. Under all ordinary conditions the secretion has a sufficient consistency to keep back the tears within the lids, but in inflammation the secretion appears to be thinner, more easily frothed, and to lose considerably in its greasy quality. With the excess of tears and the failure of the Meibomian secretion to protect the lid margins, there is an ever-increasing tendency to excoriation of the skin about the lids, and these conditions act and re-act in a vicious circle.

It therefore behoves us to be watchful of the after-conditions of the conjunctiva and eyelids of children who are recovering from measles, lest an acute inflammation of the eyelids pass on to a chronic and refractory blepharitis.

Errors of Refraction.

Excessive blinking is a phenomenon commonly seen in those whose vision is defective. Their subjective sensations are referred to a mist before the eyes, and the unconscious effect of this is an attempt to clear the eyes by rapid blinking. More tears are secreted than usual, and this excessive secretion, with the excessive rate of blinking, tends to set up a catarrh in the Meibomian glands. Let any one examine the eyelids of children at school before and after the occasion of a lesson in needlework, and he will find that a certain proportion of children whose eyelids looked normal before the work was begun, will, at its close, show pink rims to their eyelids. Now comes in the condition of excessive dirt. There are enough organisms about the eyelids of even cleanly kept persons, but when the subject is dirty, and there is an abnormal excitability of the eyelids from previous inflammation of the conjunctiva or on account of an error of refraction, the frequent rubbing of the eyes with dirty hands brings numerous septic organisms into contact with the lids, and fresh seed is sown which raises a plentiful crop of ulcers of the margins and pustules within the follicles of the lashes.

Treatment.

Realising these chains of events in the causation of blepharitis we come to a rational line of treatment.

First, the accumulations of crusts and dirt about the lashes must be removed. A solution of bicarbonate of soda of 5 per cent. strength is effective for this purpose. The solution has no bacteriological action, but it is an excellent solvent of these crusts, which are a mixture of grease, dried serum and bacteria. In ordering it for use I always direct that the lids be rubbed with wool soaked in the solution until the lashes are quite free from crusting, and until the scabby surfaces bleed. This latter direction is given because I find that the ordinary run of mothers stop their cleansing operations at the first sign of blood, and to insist upon their drawing blood is to secure a thorough cleansing of the ulcers. When the lids are clean we can effectively apply our chosen bactericidal agent. For this purpose an ointment containing some form of mercurial salt is preferable. For the most part I use the yellow oxide of mercury of 1 per cent. strength; in particularly dirty cases a dilute white precipitate ointment of the same strength is to be preferred.

These means may be relied upon to cure with expedition those cases which depend for their onset on acute causes. They will not be efficient in those which are produced by a cause which is continuous, such as a defect of vision. Hence it follows that in any case where we are not convinced of some previously acting acute inflammation, such as an attack of an acute specific fever, or where proper measures for cleansing the eyelids do not reduce the inflammation, we must investigate the refraction of the eye, and correct any error that may be present. Nothing is more striking and nothing is more satisfactory than the manner in which a long-continued and disgustingly unsightly state of sore lids in a child or young adult can be controlled and ultimately cured by the wearing of proper glasses. Obstinate inflammation, ulceration, and the gradual loss of the eyelashes will be checked, and with the resumption of more natural conditions a new growth of healthy lashes will appear as a result of the removal of the continued irritation of defective vision.

Glasses are disliked by many, and doubtless they are no addition to personal beauty, but there can be no hesitation in the choice between a pair of spectacles and the constant spectacle of sore eyelids.

Gouty Conjunctivitis.

This is another form of sore eyelid, which, when it has arrived at its full development, is no less unsightly in appearance than blepharitis. It is always chronic, and so quiet is it in its beginning, and so slow in its development, that it is almost impossible to say when it commences and how long has been its duration. As I said at the beginning there is a great contrast in this form of sore eyelid to that of the common blepharitis. Gouty conjunctivitis is almost always a disease of adults, and it is most often found to affect the well-to-do and the over-fed. The disease is not a merely local condition such as is blepharitis, it is one of the exhibitions of a general gouty condition.

For the most part the subjects of the disease have "gout" written large on their countenances; they have florid or "beefy" complexions, their tissues show a marked tendency to chalky deposits, concretions will be found in the Meibomian glands of the eyelids, in the edges of the ear cartilages, and about the joints.

The conjunctival symptoms are not continuous in the earlier stages, but are expressed by slight, evanescent, and often repeated attacks of cartarrh, and these attacks usually follow the indulgence in social festivities and the late hours associated with them. When we examine the conjunctiva we find an increased vascularity most often confined to the lower lid and the lower ocular segment; there is but little or no discharge. Examination of the lacrimal sac shows that it is healthy, so that we have not to deal with that sort of intermittent conjunctivitis induced by disease of the lacrimal sac. The patients frequently complain of itching of the eyes. The symptoms pass off quickly, even without treatment, but they reappear as speedily through many years. After many years' duration there follows a more chronic condition which simulates sore eyelids; for the chronic irritation of the conjunctiva causes a pouting of the lower lid, both by reason of the thickening of the sub-conjunctival tissue and on account of the shrinking of the skin of the lid.

Ætiology.

Before we can attempt the successful treatment of this form of sore lid it is necessary to understand the pathological processes underlying it. This is a disease produced by an irritant, but the irritant is not a living organism, it is a chemical irritant. In many cases I have examined the slight discharge by the usual bacteriological methods, and the general results have been to show that the flora of these conjunctivæ does not differ from that of the normal healthy conjunctiva; indeed, in many cases organisms were rather less frequently found. This finding does not, of course, refer to those cases where the disease has produced an ectropion of the lid and a chronic exposure of the mucous membrane, for then dust and dirt lodge on the moist ledge of the everted lid, which forms a convenient place for the cultivation of stray organisms.

In our recognition of the division of labour existing in the tissues of a highly organised animal, we lay so much stress on the resources of the kidneys and the intestines for the elimination of waste products that we are inclined to overlook the part played by the surface tissues in this important work. The skin does its share; and the conjunctiva not less, but rather more, for it retains many of the features of the primitive mucous-secreting capabilities of the primitive vertebral surface epiblast. The part played in elimination of waste products by the conjunctiva cannot be better emphasised than by calling to mind two phenomena that may be observed in the administration of drugs. Arsenic administered for a long period is apt to produce just this form of irritant conjunctivitis, for the drug is eliminated in part by the conjunctiva. Salts of iodine are similarly eliminated by the conjunctiva—a fact which must always be held in mind in the local use of mercurial preparations for the treatment of eye conditions; forgetfulness of this incompatibility of mercurials locally and iodides internally has in not a few cases been the cause of a severe attack of conjunctivitis, for the irritating iodide of mercury has been formed on the conjunctiva to the patient's and the surgeon's great discomfiture.

When the waste products of the body are excreted by the conjunctiva in excessive quantity or in qualities that are irritating, then symptoms of gouty conjunctivitis are set up.

Treatment.

It follows from this exposition of the causes of the disease that we can hope to do no good by the exhibition of antiseptics or astringents, for these

drugs are even in their mildest forms of efficiency irritating. What I have found of service has been a combined local and general treatment: The local condition has been relieved by the frequent and plentiful irrigation of the conjunctiva by a weak alkaline solution, bicarbonate of soda of 1 per cent. strength in water. And the reduction of the irritating qualities of the waste matter to be eliminated has been secured by diluting it within the body by the copious imbibition of water. It goes without saying that late hours, dinner parties, smoking concerts, and such other stimulating and irritating functions must be prohibited so far as the patient can tolerate.

Such patients as these benefit greatly by a "gout cure" at some continental spa. There may be no particular virtue in the water or the air of the spa; but in such places these people rise early, get to bed early, and drink freely of a water which, whatever else it may contain in salts, as the analysis will declare, contains H_2O in proper combination.

In those cases where the condition has persisted so long that an unsightly ectropion has been produced it is not so easy to secure a remission of the symptoms, for the conjunctiva is permanently thickened, and will not regain its normal lissomness. Then some operative procedure is necessary to replace the lid. Snellen's suture was at one time a very favourite operation for this purpose. A length of coarse silk was armed with a needle at each end; first one needle was thrust through the conjunctiva of the lower lid deep into the fornix, and brought out on the cheek below the margin of the orbit, then the other needle carried the other end of the silk through the lid on to the cheek beside its fellow end. The two ends of the suture were then tied tightly together over a piece of narrow drainage tubing, and in consequence the conjunctival fornix was drawn deeply down and the lid margin turned in against the globe. The success of the operation depends upon a species of mild seton action taking place along the lines of the suture and producing a band of scar tissue so as to permanently anchor the conjunctiva in its depressed position. With sterile silk and sterile needles the operation is less effective.

Another method, and one that is more often practised nowadays, is to dissect off the conjunctiva from the exposed part of the lower lid, and then cover the bared surface by drawing up the healthier conjunctiva and stitching it to the lid margin. Whichever mode of remedying the deformity produced by the disease be adopted, there still remains the necessity for attention to the general condition of the patient's health, lest there be a relapse of the conjunctivitis due to the excretion of irritant waste products and a return of the unsightly sore lid.

January 18th, 1909.

WITH MR. BLAND-SUTTON AT MIDDLESEX HOSPITAL.

VI.

GENTLEMEN,—I concluded the last lecture by briefly referring to the sex cell or the ovum, which I described to you as probably the most remarkable epithelial cell in the body. To-day I propose to give you some idea, from a pathological point of view, of what a remarkable cell it is. Indeed, I will go so far as to say that the most wonderful thing that man ever found out about himself was when von Baer, in 1829, discovered the human ovum or egg, and thus established the nature of the ovary, and laid the foundation for a scientific knowledge of that wonderful science which is known as embryology. Although we know absolutely that man, like all other animals, is developed from an egg, yet no one has ever seen a human egg fertilised, and no one knows anything about the changes which take place in that egg for the first fourteen days after fertilisation. All that we know about the changes which happen during the first fourteen days after impregnation is an inference from observation on the eggs or ova of birds, and some of those low sea forms such as sea urchins and star fish. After the egg leaves the ovary, and, if the environment be favourable, it will be fertilised and transformed into an *oösperm* or *zygote*. When we chance to meet the oösperm some fourteen or eighteen days later it presents a most extraordinary change, for not only has it increased enormously in size, but its investing membrane has become shaggy in consequence of the growth of these remarkable processes (Fig. 1), known as chorionic villi. In the early state the chorionic villi grow over the whole of the external membrane, but later they become collected towards one pole. In their early stages these chorionic villi are devoid of blood-vessels, and consist of an axis of delicate connective tissue covered with a layer of cells with definite cell boundaries, known by the name of Langhan's cells, and over these there is another cellular layer without definite cell boundaries, which looks like a large multinucleated cap of protoplasm lying on these cells. This is sometimes termed the syncytium, and the whole of this double epithelial cap is

known as the trophoblast, because by means of it the embryo derives nourishment from the maternal tissues. It is from these villous elements that the placenta is produced. Many years ago there was a most strenuous discussion carried on amongst the physiologists, and those who are interested in the development of the placenta, concerning the source of these two layers of cells. Some argued that the under-layer was derived from the ectoderm of the fœtus, and the outer-layer came from the epithelium which covers the decidua in the uterus. Many years ago, when I began to work at the pathology and the histology of extra-uterine or tubal gestation, I satisfied myself that no decidua ever formed in the tube. And yet we had these two layers of epithelium on the chorionic villus. Therefore it is out of the question that they could

FIG. 1.—A human embryo of the sixth week, showing the chorionic villi. (From the author's book on 'Diseases of Women.')

have been derived from decidua. But so much work has been done on this question in recent years that there is no doubt in the minds of those whose opinion is most worthy of consideration that these two layers of cells are of fœtal origin. At first the chorionic villi are avascular, but early they are vascularised through the intervention of an interesting organ known as the allantois, which later shrivels to a cord, except the part in relation with the cloaca, which becomes a receptacle for urine, and is known as urinary bladder. So that Nature is as niggardly in bestowing rewards as the State is in regard to old soldiers, for you may remember that they merely receive a small pension, which many eke out by looking after urinals and water-closets at railway stations and clubs. The chorionic villi grow with great rapidity, and embed themselves into any tissue with which they come into contact, and ultimately form that remarkable temporary organ which is known as the placenta. This is an organ which extracts, as you know, oxygen from the mother's blood, which bathes these villi and converts the blood of the fœtus into arterial blood, which then returns and supplies the fœtus with oxygen. And more than that, it is believed, and I think with truth, that these cells help the villi to extract a sort of pabulum from the blood, which is also used to nourish the fœtus. With abstruse and physiological questions of this sort I am not concerned to-day, because I want to draw your attention to one of the most remarkable forms of tumour which affects the human body.

It has been discovered in recent years that

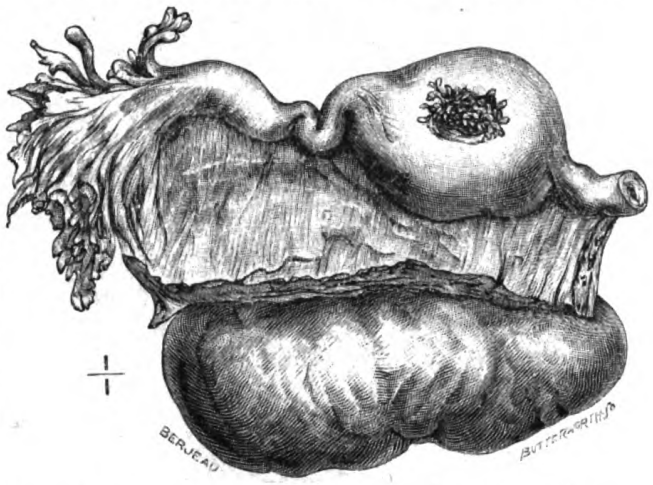

FIG. 2.—A gravid Fallopian tube and ovary. The chorionic villi eroded the walls of the gestation sac and caused fatal bleeding. (Museum of St. Bartholomew's Hospital.)

these cells are not only useful in that they form, as it were, a respiratory organ for the fœtus whilst it is in the uterus, but they also possess a very powerful property by which they embed themselves in the maternal tissues. The trophoblast possesses the power of eroding or destroying any tissues with which it comes into contact. Cancer-cells have the same power. When the cancer-cell infects an organ and begins to grow it can erode and destroy healthy tissue with which it comes in contact. Thus here in the trophoblastic tissue of the chorionic villi we have a physiological type of that erosive or invasive action of the cancer-cell. In order to give you an idea of the way in which the epithelium of the villi erode tissues and embed themselves into the underlying muscular tissue, I ask you to

look at this drawing. It is a Fallopian tube and ovary which is preserved in the museum of St. Bartholomew's Hospital, which was obtained from a woman admitted into the hospital in the seventh month of her eighth pregnancy. After she had been in the ward a little time she was seized with signs of internal hæmorrhage, and in something like eighteen hours she was dead. At the post-mortem examination they found a curious lump in the Fallopian tube, which was mistaken for malignant disease, and a small hole appears in the side of the tube through which some delicate processes protrude and float in the fluid in which the specimen is preserved. These floating processes are really chorionic villi. It is clear that this woman

Fig. 3.—Hydatid mole. (After Bumm.)

was unfortunate enough to conceive in the Fallopian tube as well as in the uterus, and when the ovum, or oösperm, as the fertilised egg is called, developed its chorionic villi, the activity or its trophoblast was so great that it eroded its way into the mucous membrane, and then perforated the muscular wall of the tube, and finally made its way through the serous coat, in which it opened up into the general peritoneal cavity, causing the bleeding which destroyed the patient. Under certain conditions, and we do not know what they are, these chorionic villi, instead of forming a healthy functional placenta, undergo a most remarkable change, and become transformed into a mass of things which looks like a collection of small grapes. Here is such a specimen. Here these chorionic villi undergo this peculiar degeneration, which is known as hydatidiform degeneration, because they look like the small vesicles which are such a characteristic feature of the disease which we call echinococcus disease (Fig. 3). For many years Virchow, who studied this disease, found that these grape-like bodies consisted of myxomatous tissue, and in structure resembled an ordinary nasal polypus, and it was then called colloid disease. Nobody troubled very much about these hydatidiform diseases until it was discovered that women who were subject to this peculiar hydatidiform degeneration of their chorionic villi were often the victims of a curious form of malignant disease of the uterus which very quickly destroyed them. And when the thing

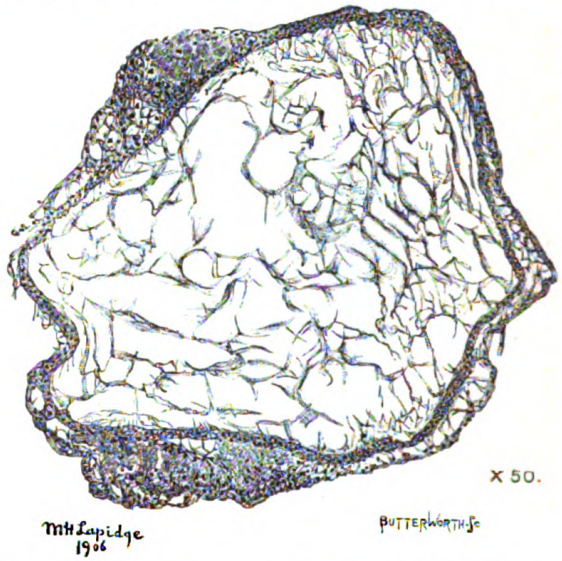

Fig. 4.—Microscopic appearance of a chorionic villus from a hydatid mole, in transverse section.

came to be investigated it was pointed out that these grape-like villi are not degenerations, but if you examine microscopical complete sections of the so-called vesicles, you find that although the centre of these grape-like bodies consists of more or less indifferent structureless tissue, yet their periphery is covered by a very active epithelium (Fig. 4). You can see that they are covered by a layer of epithelium, which in parts is extremely active, with here and there the peculiar tissue found in the decidua, and which are known as decidual cells. This was a great advance, and it was further shown by cases which have been published that after a woman has been pregnant and has had these peculiar hydatidiform changes in her placenta, some of these

vesicles have eroded their way through the wall of the uterus and stuck out on its peritoneal surface. When these hydatidiform vesicles undergo this remarkable change they are spoken of as malignant hydatidiform disease. One day it happened that Sänger, of Leipsic, had removed a woman's uterus for malignant disease, and when he made a section across it it looked as if there had been hæmorrhages into the wall of that uterus. Indeed, the cut sections of the fresh uterus appeared more like the section of pomegranate, from its colour, than anything else. And it struck him as peculiar. Therefore he put it aside and submitted it to a very careful microscopical examination, and as a result of his examination he came to the conclusion that this eroded pomegranate-looking tissue had much the same characters as the trophoblast which caps the chorionic villi. This led Sänger to describe these peculiar masses as deciduoma malignum, believing the disease to be a malignant tumour arising in the decidua. This observation was the starting-point of all we know with regard to this dreadful disease of the uterus, which later observers have shown not to be due to the decidua, but to be due to the activity of the cells which crown the villi taking on malignant changes and behaving like a cancerous tumour. And although the disease was originally named by Sänger as deciduoma malignum, he believing it to arise in the decidua, it really arises in the epithelium of the chorionic villi, and is now called chorion-epithelioma. There are two types of cells met with in this disease. In one the cells resemble this large multinuclear cell which caps the villus, and which is known as the syncytium, and the other, which is developed from Langhan's cells, and produces a much smaller type of cell. But you need not go into those distinctions so long as you remember that the disease known as chorion-epithelioma is a form of malignant disease due to malignant change taking place in the trophoblast, namely, in the syncytium and the cells of Langhan's layer.

Here is an admirable diagram showing very well the changes in connection with the villi. It is a drawing from one of Teacher's admirable specimens (Fig. 5). Here you see a villous tuft and a large collection of decidual cells with big nuclei, decidual cells with many nuclei, and here are others crowding all over the section. There is another remarkable thing, in which this growth differs from every other form of malignant disease that we know. In every other form the cells are collected together in alveoli or spaces in what is seen as strands of connective tissue, which we speak of as the stroma. In the chorion-epithelioma the tumour possesses no stroma. It consists entirely of the peculiar cells which in their usual features are identical with the trophoblast, which covers the chorionic villi. These tumours are remarkable in respect of the symptoms to which they give rise. When these tumours attack the uterus, as they do sometimes weeks after a miscarriage or a labour connected with hydatidiform disease of the villi, the patient suffers from hæmorrhages. The cells are so aggressive they

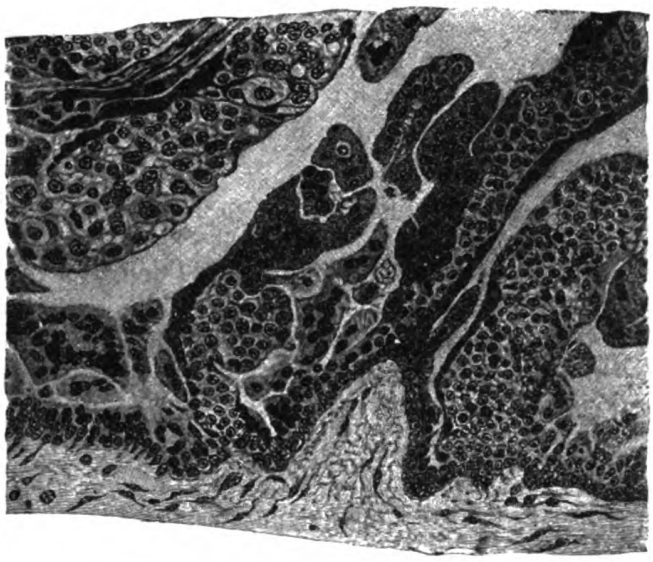

FIG. 5.—Portion of a chorionic villus from a chorion-epithelioma, showing the origin of the tumour from the epithelium of the villi. (After John H. Teacher.)

make their way through the walls of a vein and bud into its lumen, and pieces are detached and transported to various parts of the body, especially the lungs. And as I told you in the first lecture, the blood attempts to deal with these things by setting up thrombosis and destroying them. But as long as the supply continues you get the disease distributed, and if the uterus is allowed to remain in the body the patient rapidly dies. But if the disease is seen early and the uterus is removed, and the supply of malignant cells is stopped, sometimes the blood is capable of dealing with those scattered cells, and the patient may survive. Taking it altogether, chorion-epithelioma is a very remarkable disease: it is interesting as being a form of malignant disease, which has been

absolutely detected by the microscope, aided by staining methods, and it has been differentiated from that lumber-room of tumours known as malignant disease. Chorion-epithelioma is common in Germany, and fairly common in America, but it does not seem to be quite so common in this country. It is true that a fair number of cases have been detected in England, and reported to the obstetrical societies and in various papers, but, considering the large number of cases which have been published in Germany, and remembering at the same time that in this country a large number of young obstetric physicians and gynæcologists are on the look out for cases, the number of cases reported in this country is very few.

It is therefore important for you to remember that if a woman, believed to be pregnant, discharges grape-like bodies from the genital passage, she should be kept very carefully under observation, and, if she should by any chance complain of irregular uterine hæmorrhages, and particularly if she developes lung symptoms and spits blood, it is your duty to investigate her condition, and keep in mind the probability that she may be suffering from this remarkable malignant disease arising from perversions of the epithelium covering the chorionic villi. There is one other point I want to draw your attention to, and it is this: in all that I said to you about cancer, up to this point I have been trying to impress upon you all the enormous vitality possessed by the epithelial cell: it seems almost indestructible, grows on its own account, and possesses the power to do all sorts of extraordinary things. There must be something which stimulates the cell to do this; there must be a parasite somewhere. Take the sex cell. The ovaries contain some 70,000 eggs. Fortunately for the world they do not all come to maturity, but there those cells lie, and they may be shed week by week or month by month, and unless spermatozoa happens to meet them they are wasted. But if a spermatozoon gets access to an ovum the stimulus it receives from it will lead to the production of another individual. And in all these wonderful changes, apart from the production of an individual, this malignant disease may be started by the contact of one spermatozoon with that remarkable epithelial cell the ovum. It took many centuries for man to find out what it was that stimulated the ovum to form a new individual; as a matter of fact the real part played by the spermatozoon in fertilising the ovum has only been known about sixty years, need we therefore complain that it has taken much labour to find the cause of cancer? It must be a parasite, something which stimulates the normal epithelial cells of adult individuals to rapidly multiply in the same that the male gamete or spermatozoon initiates reproductive changes in the ovum. May it fall to the lot of one of you to be the lucky person to discover the stimulating agent that initiates malignant behaviour in epithelial cells.

January 18th, 1909.

Treatment of Retro-displacement of the Uterus.—Strassmann believes that this condition is more frequent than is generally believed, and that it is favoured by overfilling of the bladder, which pushes the fundus backward, and by overfilling of the rectum, which pushes the cervix forward and thus pivots the fundus backward. Another factor in backward displacement is any laceration of the cervix annulling its action as a solid tube. He urges the importance of examination four or five weeks after childbirth to detect and remedy incipient retroversion. He uses a ring pessary in certain cases, and thinks that about one fifth can thus be cured, or at least relieved from all disturbances. He warns that it is not wise to operate for retroversion resulting from inflammatory conditions in the adnexa, during the first year. The causal germs die off in the course of nine months, poisoned by their own waste products in the closed tubes. During the interim conservative measures, local application of heat, and hydrotherapy may be applied. He does not approve of operating for retroflexion unless compelled by symptoms, but adds that a spontaneous abortion should always suggest the possibility of displacement of the uterus. If a retroflexed uterus has become corrected during a pregnancy, the wearing of a ring pessary may prevent recurrence. He warns, in conclusion, that the earlier a woman gets up after childbirth the more puerperal are the genitalia, the wider the vagina, the less healed are the small lacerations which every parturient has, the larger is the uterus, and the greater the burden on the floor of the pelvis. If the woman gets up early after delivery at home, she is sure to begin to work. For these and other reasons he does not approve of allowing the parturient to get up early.—*Journ. A.M.A.*, vol. li, No. 24.

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APPENDICITIS AND ITS TREATMENT.

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I VENTURE to lay before you some conclusions which I have arrived at as the result of the treatment of over 600 cases of appendicitis upon which I have operated during the past twelve years. The comparative anatomy and development of the appendix has been explained by the great Hunter by means of five specimens which still exist in his museum. In their description he says of the appendix:

"It is a long narrow tube, about four inches in length, a continuation of the cæcum, with a definite mesentery; it is a rudiment; it is the remains of a pouch of the large intestine present in lower animals; that it remains in its fœtal state, and that in its fœtal state it corresponds with the appendix of the orang; that it often is contracted and constricted; that it contains glands and has no villi."

It is only of late years, since the researches of Sir Frederick Treves on the anatomy and position of the appendix, and the writings on appendicitis of him, McBurney and others, that the *rôle* played by the appendix in abdominal diseases has been thoroughly recognised. It is true that in the year 1826 Melier pointed out that inflammation and rupture of the appendix was the cause of acute suppurative peritonitis and of swelling in the right iliac fossa, but this was lost sight of for many years, and the cæcum has, until quite recent times, been supposed to be the prime origin of these conditions, and peri-typhlitis has been the diagnosis.

I do not propose, however, to detail the history of this affection. Let me briefly mention the causes of appendicitis: Pins, cherry-stones, bristles, fæcal concretions and foreign bodies of all kinds,

catarrh of the colon caused by wet and cold, bad teeth and imperfectly masticated food, ptomaine poisoning, the result of unwholesome food, decomposing pork, mackerel, sprats, cockles, over-ripe fruit, habitual constipation, enteric fever spreading into the appendix, and tubercular bacilli.

The morbid anatomy of this affection is, in its first stages, an inflammation of the mucous membrane, and is characterised by swelling and attended with exudation into its cavity, leading to distension. So long as the proximal end is open, and the appendix is free to move, no symptoms beyond a slight sense of discomfort and some thickening of the mucous membrane result, but should the proximal end become closed by swelling of the mucous membrane, the symptoms of appendicular distension (pain and tenderness) become manifest. Should the pressure within be able to overcome the block at the proximal extremity, the contents of the appendix are discharged into the cæcum, and the attack subsides, leaving the proximal extremity possibly a little strictured. Should, however, distension continue, and especially when round a fæcal concretion, there has been formed an ulcer in the mucous membrane of the appendix. Peri-appendicular peritonitis, perforation and rupture of the appendix will ensue, and the pus be discharged into the peritoneal cavity.

It has been clearly proved by Treves that the appendix and the cæcum are surrounded by peritoneum, and are free in the peritoneal cavity. It follows, therefore, that any exudation from within the cavity of the appendix, or any pus, the result of peritonitis around the appendix, must of necessity be intra-peritoneal in position in the first instance. The direction which the pus will take will mainly depend on the position of the appendix. It will be received, in the one case, (1) into the angle between the ileum and cæcum, and may be enclosed in omentum; or (2) it will find its way upwards and inwards, and be enclosed amongst the intestines near the umbilicus; or (3) it will drop into the iliac fossa, there to be encysted or become diffused; or (4) it will trickle over the brim of the pelvis and partially or completely fill the pelvic cavity; or (5) it will pass upwards behind the cæcum into the right lumbar region, extending upwards to the posterior region of the liver, or, occasionally, even to the under-surface of the diaphragm.

The following may be taken as the usual clinical type of appendicitis:

A young man, or middle-aged adult, enjoying his usual health, or who has been suffering for some little time previously from vague digestive troubles, is suddenly seized with acute pain in the right iliac fossa. This pain is accompanied by more or less violent colic, and is often followed by one or two attacks of vomiting. The colic passes away, but the pain remains fixed, being usually limited to the iliac fossa. The muscles of this region are hard and tense, and resist any deep exploration by palpation. There may be but little fever. From the second day the tension and the iliac pains may progressively diminish and ultimately disappear, or at the end of twenty-four or forty-eight hours a fresh crisis of general abdominal pain, with vomiting more or less incessant—of bile first, and then of intestinal contents—develops. The abdomen becomes universally hard and tense, and extremely tender. The face becomes drawn, the extremities cold, the vomiting continues, and the patient may, in a minority of cases, die of collapse, due to acute sepsis from diffused peritonitis. In other cases, the pain and tenderness, which is at first general, becomes concentrated in the right iliac fossa or elsewhere, and at the end of some days we may be able to define a more or less widely spread deep-seated tumour, over which there will still be some rigidity of the muscles of this region. The temperature, which may have been a little raised till now, is increased, and becomes remittent, with evening exacerbations. There is now, perhaps, no doubt that a purulent collection exists in the right iliac fossa, pelvis or loin.

It will thus be possible to differentiate the following definite types of appendicitis:

(1) Simple distension.
(2) Acute distension and inflammation.
(3) Rupture, with abscess.
(4) Diffused abscess.
(5) Localised abscess.
(6) Pelvic.
(7) Retrocæcal.
(8) Iliac.
(9) Recurrent.

These types are essentially distinct; each of them must be treated on its own lines. An exact diagnosis of the condition is essential for successful treatment.

It will here be convenient to enumerate and for a few moments to consider some factors which may be at work in producing these various results: (1) The position of the appendix; (2) the length of the appendix; (3) the integrity of the omentum; (4) the virulence of the bacteria; (5) exhibition of purgatives; (6) treatment by rest; (7) the health of the patient. The most important factor in determining the particular type which the disease takes is undoubtedly, in my opinion, the anatomical *position of the appendix.* There are four common positions in which the appendix may be found.

In 17 per cent. it will be found curled towards the angle between the lower end of the cæcum and ileum, or secondly, it may point, in 13 per cent., upwards towards the spleen, lying in front or behind the ileum. In both these positions (30 per cent.) the pus will find its way amongst the intestines, near the end of the ileum, and will give rise to diffuse suppurative peritonitis, which will become encysted in all probability if properly treated. Thirdly (41 per cent., Testut.), the appendix will hang free into the pelvic cavity. When the appendix ruptures under these circumstances the pus finds its way into Douglas's pouch. And lastly, in 27 per cent. of cases, it will be found to lie behind or to the one or other (usually the outer) side of the cæcum. In these cases the pus will be localised behind the cæcum, which rapidly becomes adherent to the abdominal wall. Such an abscess, when large, will pass upwards behind the ascending colon and may reach the liver, or if it still continues to increase in size it may perforate the diaphragm or may rupture and set up general peritonitis. Another anatomical factor of importance is the *length of the appendix.* The appendix may vary in length from 1 to 8 in., and it is obvious that if a tube 8 in. in length is distended with virulent pus, the pus will less easily become encysted, and will produce more extensive peritonitis than the pus from a cavity 1 in. in length. In this connection it may be mentioned that after each attack of inflammation of the appendix the tendency is for the cavity to become constricted in parts and generally shortened.

Another anatomical factor tending to modify the progress of an attack of appendicitis is the *length and integrity of the omentum.* The function of the omentum, from a surgical point of view, is to protect the general peritoneal cavity from invasion, owing to perforation of the intestinal walls. The inflamed appendix, especially one which is threatening rupture, is usually found surrounded by adherent omentum if the omentum is long enough. The longer the omentum the better can it encase the appendix or any collection of pus, however large, which has been extravasated from the appendix. I have on five occasions, by operation, found an abscess wholly encysted in omentum, which was adherent to the appendix by the thinnest possible adhesion. In one case the omental mass had become quite detached from the appendix.

Still another factor is the *virulence of the bacteria* present in the extravasated matter. It is certain that the *Bacterium coli communis* varies in virulence under different circumstances. It is obvious that a small collection of fluid containing very violent *Bacterium coli communis* will give rise to just as large an area of peritonitis as a larger collection of fluid containing bacteria of a more benign nature. The peritoneal and constitutional disturbance, therefore, other factors being equal, will vary directly as to the virulence of the particular bacillus.

Yet another determining factor is the *exhibition of purgatives,* so frequently given by anxious relations early in the case. It is obvious that one of the chief factors in bringing about the localisation of the pus extravasated from the appendix is immobilisation of the intestines, and Nature herself encompasses this by the rigid contraction of the abdominal wall, reflexly induced by the peritoneal inflammation. Exhibition of purgatives, by increasing the peristalsis, keeps the intestines on the move and prevents their adhesion to each other, and so hinders the localisation of this pus. Not only this, but purgatives, by increasing the fluidity of the fæces in many cases, are responsible for sudden extravasation of fæcal contents into an abscess cavity, and for an increase in the gravity of the symptoms.

Yet one other factor—*want of rest on the part of the patient.* A strong, healthy man has an attack of appendicular distension. The appendix is surrounded by omentum and the pain is slight. He goes to work, he strains, he detaches the omentum, ruptures the appendix and the pus is discharged. He walks home, or continues his work, and the pus gradually trickles everywhere amongst the intestines, giving rise to acute diffused suppurative peritonitis. The vomiting becomes incessant, and too late he is admitted to hospital

to die, whereas, had he gone home and to bed when first taken ill, he might have recovered.

The last factor, and one of the most important in the limitation of the disease, is the health of the patient himself. The more vigorous the patient, the more vigorous are his white blood-cells, and the better is he enabled to form an impenetrable barrier round this extravasated pus, and to localise it as an encysted abscess. Such may be their vigour that, given rest and time, frequently an abscess, which at first is diffused, will first become localised, or adherent to the abdominal wall, and may then become absorbed, or, in a few instances, may be even discharged spontaneously through the vagina or rectum. I remember quite well that in February I was asked by Dr. Aitken, of Ilford, to see a man, æt. 25 years, an athlete, who six weeks before had been suddenly taken with pain in the right iliac fossa, and had presented the usual symptoms of appendix inflammation. The temperature rose, and a lump in the abdomen gradually formed. The temperature fell, and the pain subsided. After this, on three separate occasions he had, during the ensuing six weeks, exacerbation of pain with a rise of temperature, and an increase in size and tenderness in the tumour. When I saw him he was thin, emaciated, his temperature 101, and his pulse weak. He had a large, deep-seated, movable, globular swelling, about five inches in diameter, situated immediately behind the umbilicus. It was only by careful palpation that I could define a narrow line of dulness extending from the iliac fossa into this tumour, and so, guided also by the history, was enabled to make an exact diagnosis. I operated, found the intestine adherent to the abdominal wall in the iliac fossa, insinuated my finger between the adherent intestines, and so reached the abscess, inserted a tube, and he recovered. On four distinct occasions had this abscess burst its bounds and become diffused. Had his white corpuscles not been in splendid condition he must have succumbed.

The above are, I think, the principal factors concerned in the determination of the course which a given case will pursue. In every case it will be first of all necessary to decide whether the patient has appendicitis or not. If there be a history of sudden pain in the right iliac fossa, or about the umbilicus, with resistance in the iliac fossa, and localised tenderness at one spot, a pulse of 100 to 120, a temperature of 100 to 102° F., nausea or vomiting, and a history of constipation, we may infer that the appendix is inflamed and distended. It will often be possible, even in the early stages, to make out a small oval patch of dulness in the right iliac fossa, corresponding with the distended appendix, and the diagnosis will be confirmed if there has been a previous attack similar in character. When, with the above history, there is a well-defined tumour in the right iliac fossa, or if the abdomen be rigid and acutely tender, especially in the right iliac fossa, the case is almost certainly one of appendicitis.

It is not, however, always possible to make a certainty of the diagnosis, especially in the early stages. There are many conditions with which an attack of appendicitis may be confused, of which the following are the most important: Gall-stones, duodenal catarrh with gall-bladder distension, ovaritis, pelvic tumours. In *gall colic* and *duodenal catarrh* the pain may also be sudden and severe, and accompanied by vomiting, but in these conditions the pain is situated in the gall-bladder region, where, it must be remembered, the appendix, owing to non-descent of the cæcum, may also be located. The pain of *ovaritis* is also iliac, but will usually be on both sides of the abdomen. The tumour due to a distended appendix, with an abscess enclosed in firm adhesions, sometimes very closely simulates malignant growth of the cæcum. In both you have wasting and vomiting, and in both the tumour may be nodular and irregular in outline. In both there is pain, but in carcinoma the pain is long continued, and the tumour slowly increases in size, and has been slowly formed, whereas in appendicitis the pain tends to decrease, and the tumour to become smaller in size. Distension of the pelvis with pus (following a ruptured appendix) may simulate distended bladder, or a pelvic abscess or tumour from other causes. Retro-hepatic appendicular abscess will simulate an ordinary liver abscess or tumour and an empyæma. It will also be necessary to distinguish between a ruptured appendix and a perforated duodenal or gastric ulcer. In both there is general peritonitis, and may be persistent vomiting. In both there will be a history of digestive trouble, and in many cases of appendicitis the pain is epigastric in the first instance. Time does not suffice to enable me

to enter more fully into the question of differential diagnosis.

Here a few words about McBurney's point, and two other aids to diagnosis.

First, I cannot say that I have found McBurney's point as reliable as other people are said to have done. It is rare, in my opinion, that McBurney's point corresponds to the position of a distended appendix or an abscess round the appendix. If the abscess be behind the cæcum, it is above, and external to McBurney's point. If the appendix hangs down towards the pelvis, as it does in 41 per cent., or even where it is curled up somewhat upon itself, the abscess is, at least, one inch below and in front of the line joining the anterior superior spine with the umbilicus, in which line, at the outer edge of the rectus, McBurney's point is situated.

Secondly, another aid to diagnosis is the superficial tenderness of the abdominal wall, reflexly excited by intra-abdominal irritation. My colleague, Dr. Head, has pointed out that in early appendicitis there is, lying round the abdominal wall just below the umbilicus, a line of superficial hyperæsthesia, and later on, as more and more of the peritoneum becomes inflamed, this area of hyperæsthesia extends. It follows, therefore, that so long as this definite line is a narrow one, we may infer that the condition is local, and that the appendix is simply distended.

Thirdly, the third aid in diagnosis is the leucocytosis test. Usually, of course, the local symptoms will enable one to decide as to operative intervention. This is especially true when the local symptoms are very acute. On the other hand, in those cases of appendicitis in which the local symptoms are less marked, and are insufficient to enable a positive diagnosis to be made, I have in several instances found the count of the leucocytes of value. It is a guide to the presence of pus, but not to the amount of it. The number of leucocytes rises in the early stages of the disease, and begins to fall when the patient is recovering, or should he be about to die. It is also in the more chronic condition of appendix trouble which simulates tubercular peritonitis that the leucocytosis count is of great value. In cases of fulminating appendicitis, however, the toxæmia may be so rapid and so severe that the organism becomes extremely depressed, and may fail to manufacture leucocytes. The number of leucocytes in the blood, under these circumstances, may even be subnormal. The leucocyte test, therefore, is useless in the very case in which we most need aid in diagnosis.

We will take for granted that the case is one of appendicitis, and we are called to it during the first twenty-four hours of the attack—what are we to do? We must agree with American surgeons, whose standpoint may be stated as follows:

"First, that it is often possible to make an absolute diagnosis of appendicitis within the first twenty-four hours; that at this period the infective material is still circumscribed by the wall of the appendix; that within another twenty-four hours it is impossible to tell what the pathological condition will be, and impossible to predict whether a favourable or unfavourable termination will supervene; that the pain and pulse and temperature can never be absolutely relied upon as guides to the condition of the parts in the right iliac fossa, and that, therefore, an exact diagnosis is impossible. The pain may have disappeared, the pulse may be 84, the temperature 99° F., and yet the patient's appendix may be completely gangrenous without adhesions. A gangrenous appendix gives rise to no pain, it does not become encapsuled, nor does it undergo absorption. It is a septic foreign body which may give rise to spreading peritonitis." I agree that if the case is seen within the first twenty-four hours and the diagnosis certain and the symptoms are at all acute, the appendix should be removed at once.

It must be remembered, however, that the majority of cases of appendicitis are not seen until after the first twenty-four hours have elapsed, and after that time it is impossible to say that the appendix has not given way. On the other hand, the American practice of immediate operation in every case prevents subsequent attacks, with the loss of time and danger to health consequent thereon, and that this is somewhat considerable let the case of G. F— speak.

G. F—, æt. 11 years, was admitted under my care at the London Hospital in April. There was a history of irregular pyrexia for several weeks and an abscess in the right flank. This was opened and drained. The wound granulated slowly, and he went out with a sinus. On June 11th the boy was re-admitted for appendicectomy. The abdo-

men was opened and the cæcum was found very adherent to the abdominal wall, and was accidentally opened. It was sutured and the idea of removing the appendix was given up. The wound healed by first intention. In September the boy was re-admitted with another large abscess adherent to the abdominal wall, which was opened and drained, and he made a good recovery. In the following February the patient was again admitted, this time with the pelvis full of pus. This was opened; it drained well and closed. Subsequently his appendix was removed, and he was discharged cured. Had he been seen and the appendix removed during the early period of his first attack he would have been saved almost a year of continuous illness.

The English point of the question, from a medical point of view, was well put by Dr. Hawkins, and may be summarised as follows:

"It appears probable that by a routine excision of the appendix at a very early period some few cases might be saved from a general peritonitis, but for my own part I doubt whether the gain of life is, on this plea alone, large enough to counterbalance the disadvantages of the wholesale surgery which such a practice would entail."

My own opinion is as follows:

I think it possible in the majority of instances, when the case is seen early, to infer with tolerable certainty that the appendix is simply distended. In such cases, if the pain is not severe, with little tenderness over the appendix, with little or no vomiting, little or no risk of temperature or pulse, I would treat the case medically, but if (as happened in a case I remember in consultation with Dr. Hoar, of Sidcup, within twelve hours of the onset) the pulse is very rapid and is increasing in rapidity, the patient is restless, the abdomen is rigid, there is vomiting, and especially if on percussion of the right iliac fossa a localised, small, oval patch of dulness and tenderness is discerned, immediate operation should be performed. The patient above mentioned, six hours after I saw her, suddenly felt very severe pain all over the abdomen and became collapsed. The pulse increased in frequency to 140 and the temperature fell; I have no doubt that up till that moment the appendix was acutely distended only, and at that moment it ruptured, causing diffuse septic peritonitis, for which I operated without relief. The case was one of true fulminating appendicitis. When therefore, the pain is very severe, the pulse above 120, and distension of the appendix is inferred from the presence of a *small* localised patch of extreme tenderness on pressure, and of dulness on percussion with abdominal resistance over it, within twenty-four or thirty-six hours of the onset of the disease immediate operation is imperative. Other cases where the symptoms are mild should be treated as follows:

The *pain must* be allayed with warm applications, with opium, to the abdomen. For the *nausea and vomiting* ice may be given to suck. It is chiefly on the sensitive, painful spot in the right iliac fossa we should fix our attention. If the fixed pain continues but little marked and the abdominal wall is relatively pliant we may continue the fomentations. If, however, the pulse increases, the temperature continues to rise, if the tenderness increases, if the general condition of the patient tends to get worse, operation must be resorted to without delay. The exhibition of opium is absolutely essential. It allays the pain, and diminishes peristalsis, and this favours the localisation of the pus, should the appendix have ruptured. Many surgeons advise against the administration of opium, because it masks the symptoms, but a dose of opium may make all the difference between life and death to a patient. How, therefore, can we justifiably refrain from its exhibition, because possibly at some future date the opinion of a consultant may be biassed? In any urgent case, therefore, the second opinion ought to be summoned early, but a dose of opium should be given immediately. It is often not necessary to give opium for more than a few days. Rest in bed is absolutely essential. The confinement to bed should be continued for some time after the cessation of all local pain. The diet should be fluid till the temperature is once more normal. Calomel or an enema should be given, but no active purgatives till the symptoms have quite subsided. By this treatment the majority of attacks will subside, and the appendix may then be removed during the quiescent period.

Should the case be acute, and operation be determined upon, an incision must be made two to three inches in length parallel with Poupart's ligament, an inch from the anterior superior spine; the fibres of the external oblique must be separated

and retracted; the fibres of the internal oblique are separated and those of the transversalis muscle and fascia cut. These are retracted with hooks, so that a gridiron aperture one inch square is formed. The peritoneum is exposed. It is picked up with forceps, and a tiny opening is made, into which a blunt-curved bistoury is passed, and the aperture in the peritoneum is enlarged sufficiently to admit the finger. The appendix, if free, is easily withdrawn through this aperture by the finger. Should there be adhesions, these must be gently broken down by the finger, and the appendix withdrawn.

If it is necessary to withdraw the cæcum, it should be returned to the abdomen, if possible, as soon as the appendix has been exposed. The proximal end of the appendix should be found. A Spencer-Wells forceps should clamp it three-quarters of an inch from the proximal end. The peritoneal coat should be divided circularly on the proximal side of the forceps, and a coat-sleeve of peritoneal covering should be turned upwards towards the cæcum exposing the mucous coat. This should be clamped, tied with thin silk and cut through, the sleeve of peritoneum turned down to cover the mucous stump and its end sutured with fine silk. It is not necessary to cauterise or scrape the end of the mucous tube; it should be washed. The mesappendix is then exposed and ligatured in two parts with stout catgut. The appendix is next removed. The abdominal wall is satured with catgut in separate layers. No tube is necessary. The skin should be sutured with silkworm gut. Antiseptic dressings should be applied.

Let us now proceed to consider other types. We are called to see a patient whose face is pinched and anxious. He is restless, and a little cyanosed. He vomits more or less continually, especially on being moved, his abdomen is distended, rigid, and tender, especially at the lower part and at the right iliac fossa. He can only rest in one position. From the history one learns that he has been suddenly taken with pain in the right iliac fossa, perhaps two days previously. The pain persisted and suddenly got worse, and the vomiting became incessant. The diagnosis is one of ruptured appendix. The question is, Where is the pus? In the iliac fossa, or in the pelvis, or behind the caecum, or in the general peritoneal cavity? It is absolutely essential that the exact location of the pus should be made out. No blind haphazard exploration of the abdomen is good surgery. An exact diagnosis must be made. For this purpose, very gentle and careful palpation of the abdomen must be made.

Pelvic Abscess.

If at the lower part of the abdomen some slight impairment of resonance or possibly absolute dulness be perceptible, if the upper limit of this dull area is a line running more or less transversely across the lower part of the abdomen above the pubes, if also there be some resistance on palpation over this area of dulness, we must suspect that the pus has found its way into the pelvis, and has caused pelvic peritonitis, commencing in Douglas's pouch and spreading evenly upwards on to the abdomen, causing adhesion of the intestines and dulness on percussion. I have found this transverse line of dulness of the greatest service in diagnosis. A vaginal or rectal examination should now be made, and will reveal a soft, doughy swelling, filling Douglas's pouch, pushing forwards the bladder and uterus, and compressing, or sometimes projecting into, the rectum behind. Under these conditions, especially if there be pain on, or frequency of, micturition, pain on defæcation with tenesmus, there should be no doubt that the pus has filled Douglas's pouch. If the vomiting is very severe and the patient is very collapsed, or if there is a history that he is getting worse, immediate operation is imperative. Should, however, the diagnosis be doubtful, or the symptoms not urgent, a delay of twelve hours or longer may be permitted, with the idea of allowing the pus to become more firmly encapsuled by the intestines, and its presence in the pelvis more definitely diagnosable.

The method of procedure which I adopt is as follows: The bladder is emptied; a short incision one inch and a half in length, immediately above the pubes, is made in the linea alba. The recti, etc., are separated and divided down to the subperitoneal connective tissue. When this is reached further dissection must be made to one or other side in front of the bladder for about one inch or more from the middle line, so as to reach the edge of the bladder. Having got beyond the lateral edge of the bladder the peritoneum must be opened.

Adherent to its inner surface coils of intestine are usually found. The forefinger must be passed within the peritoneum, and must gently detach the adherent intestine, travelling backwards and downwards along the posterior wall of the bladder until, usually deep behind the bladder, the finger suddenly slips into space. This is the abscess cavity. The finger is withdrawn, and a drainage-tube introduced. The incision must be made immediately above the pubes, and care be taken to avoid breaking down the adhesions above the incision, and so opening the general peritoneal cavity. In the female it is, in my opinion, well at this stage to pass along the vagina a trocar and cannula large enough to carry a medium-sized drainage-tube. With this, or with a curved bistoury, the roof of the vagina is punctured, a drainage-tube is passed into Douglas's pouch, and brought out through the abdominal incision. It is much more easy to insert this vaginal drainage-tube at the time of the operation than at a subsequent date. This tube effects very efficient drainage. It is not safe to attempt to open these abscesses *per vaginam* without having first located the abscess by the abdominal incision. In one of my recent cases Douglas's pouch was filled by a large ovarian cyst which was surrounded by pus.

The advantage of this method of drainage will be obvious from the following case:

E. B—, a servant, æt. 18 years, was admitted under my care to the London Hospital on December 3rd. There was a history of severe pain in the right side, spreading to the umbilicus, of four days' duration, and with an abscess in the appendix region, which was opened, and about 10 oz. of fæcal pus let out. The cavity was the size of an orange, and was apparently shut off from the rest of the peritoneal cavity. A drainage-tube was inserted. For two days she made favourable progress, but on January 4th an area of dulness was found in the lower abdomen, extending upwards to a horizontal line joining the anterior superior spines of the ilium. She still had much pain. The temperature was 99° F., and the patient was very restless and weak. An incision was made in the middle line above the pubes, the peritoneal cavity opened, and about 10 oz. of very fœtid pus escaped from the pelvic cavity. A trocar was passed along the vagina through the posterior fornix, and a drainage-tube inserted into Douglas's pouch. The patient made an uninterrupted recovery, the tube being removed from the vagina twelve days later.

In the above-mentioned operation, it is especially important to empty the bladder prior to operation and to work laterally before the peritoneum is opened, so as to avoid perforating the bladder. On several occasions I have accidentally opened this viscus, though it had been emptied. In each case the wall of the viscus was sutured, and the patient recovered without a bad symptom.

Retrocæcal Abscess.

Should palpation of the abdomen reveal a patch of dulness, the lower end of which is just within or just above the anterior superior spine, and which extends upwards along the crest of the ileum, being limited anteriorly roughly by a more or less vertical line, then I consider it may be inferred that the appendix lies at the back of the cæcum, and that a post-cæcal abscess exists. Such cases are not always of extreme urgency, and may safely be watched for some little time. The dulness will become more pronounced and defined, the pyrexia may become more definitely hectic in type, the patient may have diarrhœa, and the vomiting will subside. So long as the pulse tends to decrease in frequency, the temperature to drop, the tenderness to subside, the tumour and area of dulness to decrease in size, the case may be carefully watched from day to day. Should the area of dulness be very large, or should the symptoms tend to get worse, or should the tenderness be very acute, or should the temperature rise after having fallen to normal, operation is imperative. There may be a large abscess, and the temperature and pulse may be normal. An incision three inches in length should be made, one inch above and parallel with the anterior third of the iliac crest, the abdominal muscles divided, and the peritoneum reached and carefully opened. The cæcum will, in all probability, be found adherent to its inner surface. By passing the finger downwards towards the back of the cæcum, the abscess will be reached and a drainage-tube inserted. Should the abscess be large and extend far upwards, it is wise, unless a thick layer of tissue intervene between the abscess cavity and the skin of the loin, to bring a drainage-tube out at the loin. In many cases recovery by granulation will slowly be brought about. In a

few cases a sinus will persist, and it will then be necessary thoroughly to explore the sinus tract, and remove any remnant there may be of the appendix, and provide perfect drainage from the upper end of the abscess cavity through the loin if this has not been already done. If the dull area in the flank has extended up to the liver region, if there have been symptoms pointing to trouble in the hepatic region, such as pain, tenderness, jaundice, cough, and pain on respiration, the abscess cavity should be thoroughly explored by means of a probe. This will usually be found to pass upwards towards the flank. In this situation it should be cut down upon, and from this point the upper end of the abscess cavity again thoroughly explored. A large collection of pus may be found round the kidney, or at the back of the liver, or above the liver between it and the diaphragm. I have known such an abscess cavity to be filled with fœtid gas, to push up the diaphragm and to diminish the area of liver dulness, and so lead to the diagnosis of intestinal perforation. I have known such an abscess to perforate the diaphragm, and be coughed up through the bronchial tubes. This pustular expectoration gradually ceased when free drainage of the subdiaphragmatic abscess was effected. Free drainage is essential, and the cavity must be thoroughly irrigated.

For the after-treatment of these abscess cavities I have used peroxide of hydrogen as an injection with very marked benefit, two ounces being injected twice daily.

As a typical case of this condition I may quote the following:

T. W—, a tailor, æt. 46 years, was admitted under my care to the London Hospital on October 13th. There were symptoms of thirteen days' duration (pain, tenderness, vomiting) and an area of dulness in the right flank, with rigid abdomen, and pain and tenderness in the right iliac fossa. Temperature 103° F., pulse 108, tongue furred, and shallow respiration.

October 13th: Incision above iliac spine opened; a large, foul, retro-cæcal abscess found; tube inserted.

November 1st: Continued to discharge foul pus, and temperature remained irregular.

November 6th: Sinus probed and thoroughly examined; found to extend upwards towards hepatic region. Fresh opening made in loin and sub-diaphragmatic region explored; large abscess containing pints of foul pus found behind and above the liver; a drainage-tube eight inches long inserted.

November 7th: Cavity discharging freely. Temperature fallen to normal from 102° F.

December 21st: General condition remains bad, and sinuses show no sign of healing.

December 31st: During past ten days cavity has been irrigated with peroxide of hydrogen, and a marked improvement has set in.

January 14th: Patient discharged to convalescent home with sinuses healed.

Iliac Abscess.

Should palpation of the abdomen reveal a patch of dulness in the right iliac fossa, with a hard, well-defined edge of semi-circular outline, and should there be a softer centre situated just above and in front of the outer half of Poupart's ligament, very little doubt need be entertained, but in the centre of this mass there will be found one, two, or three drachms or more of pus, an encysted localised intra-peritoneal abscess. Seeing that the edge is firm and well-defined, the abscess is probably of some days' standing, and the adhesions will be of some strength. We may therefore proceed to open this by means of an incision 1½ inches in length, parallel to Poupart's ligament, and over a point a little nearer to the ligament than the centre of the swelling. The skin, the external oblique aponeurosis, and the internal oblique muscular fibres are divided from the whole length of the incision; the wound is retracted, and the fibres of the transversalis muscle and fascia are then divided for about an inch, layer by layer. With the index finger in the wound, and the hand on the abdomen, the centre of the tumour must be carefully located, and the peritoneum opened opposite this point for about three quarters of an inch in length. Should the muscular fibres be œdematous, matted together and paler than normal, and the peritoneum be whitish in colour, pus may be expected to exude immediately the peritoneum is perforated. Should, however, no pus escape on opening the peritoneum, the left forefinger must be inserted, and the gut, which will be found adherent, must be carefully detached. The finger must be slowly passed in a downward direction towards the iliac fossa, separating the soft adhesions as it goes. During this manœuvre the finger may suddenly

slip into a space, which is the cavity of the abscess. The finger is withdrawn, and the pus allowed to escape, and a tube inserted. Should no pus be found on passing the finger backwards into the iliac fossa, it will usually be found that the finger has passed beneath the mass, and it must then be carefully inserted into the centre of the mass, adhesions being very gently broken down in its progress. In the centre of the mass pus will usually be found, but not always, because the abscess may have been absorbed. Nothing but an empty cavity may be found. If the pus comes out under pressure it may usually be assumed that the pus is encysted, and a satisfactory prognosis may be given. If, however, very little pus exudes, and that not under pressure, it is probable that diverticula from this abscess cavity run down into the iliac fossa, or in amongst the intestines, and that the abscess is no longer encapsuled. Under such circumstances the question is whether it is advisable then and there to open the abdomen and explore the pelvis or general peritoneal cavity. As a general rule, however, the tumour having been defined, it is better to await the result of draining the present abscess. When the cavity is opened, should the appendix readily present itself it may be ligatured or twisted and removed, otherwise it should be left. The cavity should be very gently explored for any fæcal concretions, but on no account must the wall of the abscess cavity be roughly treated, nor is irrigation of this cavity advisable. By forcible irrigation the weak adhesions, which form the barrier to the entrance of the pus into the rest of the general peritoneal cavity, are broken down. This originates a second attack of diffuse suppurative peritonitis, which, together with the shock from the operation, kills the patient. An abscess, which has been given time to become thoroughly localised, and which is opened without irrigation, in practically every case gets well.

I have stated above that if on opening an appendix abscess the appendix presents itself it may be ligatured and removed, otherwise it should not be explored for. My reasons for not removing the appendix when opening the abscess are as follows: (1) The parts are bathed in pus; they are very difficult to define; the operation is prolonged and the risk to life increased. (2) The parts are very soft and friable; there is increased risk of rupturing the intestine and producing a fæcal fistula; there is increased difficulty in closing the proximal end of the appendix. (3) The difficulty in finding the appendix may be so great that in isolating it the adhesions which have enclosed the pus may be broken down and the pus may escape into the adjacent peritoneal cavity, giving rise to a further outbreak of acute suppurative peritonitis which, combined with the shock, may just be sufficient to kill the patient. (4) Exploration after the appendix entails a far longer incision; it must be remembered that any incision made at this time is bathed in septic pus and fails to heal by first intention, the risk of ventral hernia is materially increased by this procedure. (5) It cannot be too frequently pointed out that the appendix when found will nearly always be ulcerated and more or less friable at one point; at this point it will break away, leaving, in all probability, more or less of the proximal end still unobliterated, and which owing to inflammatory lymph may be overlooked, and in which proximal portion of the appendix another abscess may at any time supervene. For these reasons, therefore, it is not worth while increasing the risk by being too energetic in searching for and removing the appendix at the time the abscess is opened.

Subsequent Removal of Appendix.

The question whether the appendix ought to be removed at a subsequent sitting will, in my opinion, entirely depend upon the situation of the abscess. Should the abscess have been retrocæcal in situation, it is not advisable, in my opinion, to explore again in order to remove the appendix unless the discharge shows no signs of ceasing, or unless the wound shows signs of forming a fistula. Statistics show that not more than one in twenty-three persons who have had a retrocæcal abscess which has been opened and has healed up have any further trouble with the appendix. In many of these abscesses the appendix has been found wholly destroyed. Should such a recurrence of inflammation or recurrent abscess develop it may be allowed to subside or may be opened, and the appendix thoroughly explored for and removed, the risk of ventral hernia notwithstanding.

Should the abscess have been a pelvic one, in every case the appendix should be removed, because in nearly every case the appendix is hanging down into the pelvis, and is ulcerated near its distal extremity. The best period for removing

the appendix is as soon as the discharge from the pelvic abscess shows marked signs of decreasing in quantity.

Where the pus is in the iliac fossa, should the patient have had many attacks and the abscess have been a large one, and the cavity have healed completely, it is permissible for the patient to assume that the appendix is destroyed, but in every other case, and should there be any doubt, it is better that the iliac fossa should be explored and the appendix removed at a subsequent sitting. It follows, therefore, that the appendix should be left under the above circumstances in retrocæcal abscess; that it may be left occasionally where the abscess has been iliac; and that it should never be left unremoved where the abscess has been pelvic. Should it be decided to leave the appendix, it is possible that another attack of appendicitis may develop.

If possible this attack also should be allowed to subside, and at a later date the appendix must be removed. The operation is not necessarily more difficult because there has been an abscess. I have on several occasions removed the appendix from a person in whom there had been an attack subsequent to the opening of an abscess, which was adherent to the abdominal wall in the right iliac fossa. In some cases the appendix has been free in the abdomen, there being absolutely no adhesions at all round it, and in the others the adhesions have been slight.

Diffused Suppurative Peritonitis.

So far I have dealt with those cases where there is dulness, well marked either in the lower abdomen, in the right flank, or in the right iliac fossa. I now come to those cases where, with the abdominal rigidity and tenderness more or less general, the dulness in the right iliac fossa is absent or ill defined. It will nearly always be found that the abdomen is most rigid and tender in the right iliac fossa, and the question arises, Is the pus encapsuled or not? And if, guided by the fact that there is no definite well-marked margin to the dulness, we think the pus is free in the peritoneal cavity, the question is, can we afford to give the patient the chance of bringing this encapsulation about? Here the history of the case is of great service. If the man, in spite of opium and rest, is getting worse, no delay is permissible. The appendix region must be explored at once. In many cases it will be found, however, that the patient has only just been brought into hospital, that no opium has been given, that purgatives have been given, or that the patient has not been kept in bed. Or it may be stated that he is a little less tender on the left flank than he was on the day before, or we may have reason to doubt his having been placed under conditions favourable to encapsulation. Under these circumstances it is permissible to wait for a few hours, in order to give the intestines a chance to shut off and localise the pus. A few hours' delay will determine whether this is likely to occur. By that time the patient will be no worse; the left half of the abdomen may even be a little less tender and rigid. The vomiting possibly may be a little less urgent, the dulness possibly a little better defined, and so from day to day the case may be watched; the rigidity of the left half of the abdomen will disappear, and the dulness on the right half become more and more defined, though smaller in area. The temperature will fall to normal, the pulse will decrease in frequency, and the vomiting will cease. The subsidence of this lump may continue until it absolutely disappears—in other words, the pus is absorbed, or there may come a point at which it remains stationary. Should the tumour have become well defined and then remain stationary it should be opened. If at any time in the course of the retrogression of the lump the temperature rises and the tenderness over it increases, immediate operation is imperative. In many cases, however, the lump will partially or completely disappear by absorption, and a week or ten days after this event, and the temperature has become normal, appendicectomy ought to be performed. In certain cases, however, the abdomen is rigid, distended and tender all over, the vomiting is incessant, the pulse over 130, the temperature high or subnormal, the eyes sunken, the patient restless, and no area of dulness can be found on percussion. Here it may safely be inferred that septic pus is diffused amongst the intestines, and is never likely to become localised. Under these circumstances immediate laparotomy, with thorough cleansing of the abdominal cavity and drainage and continuous transfusion, is the only chance of saving the patient's life. My statistics point out the hopelessness of such cases. In 1903 I wrote of twenty cases of acute suppurative peritonitis treated by

laparotomy and drainage, in some cases with irrigation, in some without, and in some cases with removal of the appendix and some without. Twenty died. Many of them were in a hopeless condition of collapse, due to bacterial absorption, when first seen. We must look to early diagnosis of this affection, and immediate energetic medical treatment, supplemented by the prompt exhibition of the correct surgical procedure, for the prevention of such hopeless cases.

Relapsing Appendicitis.

We have yet to consider the fourth type of appendicitis, the relapsing or recurrent type.

A mild attack succeeded by another mild attack, treated medically with recovery, until one attack develops an abscess or acute suppurative peritonitis, with possibly a fatal issue. For these people removal of the appendix is imperative. It should be performed during the quiescent period after the subsidence of an attack, preferably when the temperature has been normal or thereabouts for a week or ten days. When the operation is done at this period the adhesions are not too firm to be broken down, but the adjacent parts of the intestine are no longer softened by acute inflammation. The rule I have laid down for myself is to remove the appendix after the second attack in *every* case, or after the first attack if the diagnosis is certain, or if the attack has been severe, or when the recovery from it has been slow, with more or less continued pyrexia subsequently. Under these circumstances it is fairly certain that the appendix is still distended, possibly contains a fæcal concretion, and a second attack may be expected at any time. The appendix, therefore, is best removed. A few people never have more than their first attack of appendicitis. Such people apparently make a complete recovery. It is therefore not advisable to operate in every case after one attack only. Removal is performed as follows:

Through the usual incision the abdomen is opened, the appendix located, and if possible, drawn up into the wound. If there be adhesions of a recent nature these may be easily broken down, but if they be old the incision must be enlarged so as to give the operator a good view of these adhesions, which, being divided before, the cæcum and appendix with it can be brought out into the wound. In many cases there are no adhesions, and here all that is necessary, having found the appendix, is to replace the cæcum, shut off the peritoneal cavity with sponges, the appendix being held well within the wound with a Spencer-Wells' forceps attached to its centre. A circular incision is made round the appendix through its peritoneal coat, half an inch from its proximal end, and a cuff of peritoneal tissue is turned backwards towards the cæcum. To the mucous tube a ligature of thin silk is applied, close to the cæcum, the tube is divided, it is touched with carbolic, and the cuff of peritoneal tissue is turned forwards over the end of the mucous tube, and sutured with fine silk. The mes-appendix is now ligatured in two pieces with gut, and the appendix removed, the peritoneal cavity cleansed out and the abdomen closed. Union by first intention is the rule. In certain cases, however, the appendix will be found very adherent. Great patience and skill are required to isolate it from these adhesions. When isolated it is removed as above. The worst adhesions are found in those cases where the appendix is lying behind the cæcum. The number of attacks vary from one to thirty-seven. The appendix is found obliterated or patent, three quarters of an inch or five inches long, containing fæcal concretions or muco-purulent fluid, the lumen strictured at one or more spots, perforated or gangrenous, or completely detached at its proximal extremity, adherent or free, straight or very tortuously curled.

January 25th, 1909.

Experimental Influencing of Pregnancy.—Hofbauer reports experiments in which cholesterin, the physiologic opponent of lecithin, was fed to gravid animals. He gave the cholesterin added to the food. In every case there were destructive changes in the placenta, evidently the result of the action of the cholesterin on the lecithin which abounds in the placenta. The changes in the placenta resembled in every respect those observed in syphilitic placentæ. This suggests the possibility of a uniform explanation of the various facts observed in regard to the action of cholesterin—the "physiologic antipode of lecithin"—in syphilis and in cancer, the tissues in the latter being unusually rich in lecithin.—*Journ. A. M. A.*, vol. lii, No. 1.

DISEASES, NOT OF THE STOMACH, WHICH ARE LIABLE TO PRODUCE MISLEADING GASTRIC SYMPTOMS.

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AMONG the many perplexing clinical problems which confront the practitioner few are frequently harder to solve or more difficult to explain than those which concern the varied derangements of the stomach. The word "dyspepsia" itself is but the expression of a condition as varied and multiple in its manifestations as the causes upon which it primarily depends. As vague as the term "rheumatism," it is often, like that expression, used in a substantive sense, and taken to indicate that what is only a symptom is really the disease, and something to be treated regardless of the true cause upon which it depends.

It would carry me far beyond practicable limits to venture upon any exhaustive discussion of the subject of dyspepsia. My intention in these few remarks is solely to confine them to some of the more striking illustrations of derangements of the stomach dependent upon disease elsewhere. In other words, to show how easily symptoms apparently, and in some cases really, gastric may mask a disease acting only indirectly in relation to the stomach.

There are many reasons why certain symptoms should be taken as esentially gastric and of primary gastric origin. Among the most cogent of these is the anatomical position of the stomach and the relation which it bears to neighbouring parts in its nerve supply. As regards its situation, it is in close proximity to the liver, gall-bladder and gall-ducts, to the pancreas, and to the duodenum and transverse colon. In its nerve supply, which is through the vagi and sympathetic, it is brought into relationship with more distant viscera such as the whole intestinal canal, the lungs, and the heart. In addition to these two considerations the influences of the circulatory system generally, and of the brain and spinal cord, are important factors which must not be left out of count.

Thus, from so brief a narration of the multiple associations which normally connect the stomach with other parts of the body, it will at once be gathered how equally numerous must be the bearings of disease situated elsewhere upon the functions of a viscus naturally prone to manifest symptoms under provocation of even a trivial character.

From abstract considerations let me now pass to concrete illustrations, and select for this purpose a few of the most misleading cases that may be met with. And first I will take—

Carcinoma of the Colon

as represented by that more common type of the tumour which slowly but gradually contracts the calibre of the canal. In the early stages of this disease, before obstructive symptoms become clearly obvious, digestive derangements are among the commonest manifestations; and these patients frequently complain of being subject to "bilious attacks." There is no marked abdominal pain, and such discomfort as is experienced is usually ascribed to flatulence, or the taking of something that had disagreed. In the eyes of the doctor the symptoms are frequently ascribed to a gastric condition, and accordingly stomachic sedatives are administered, and a careful attention to diet recommended. The result is usually good, and the patient, for the time being, is relieved and improves. However, as time goes on another attack ensues, accompanied with a little more abdominal pain, less inclination for food, and a greater tendency to nausea and vomiting. Still further significant features appear, such as gradually increasing weakness, and an insidious diminution of body weight.

The nature of the symptoms, both as regards severity and frequency of occurrence, is considerably affected by the situation of the growth; and perhaps the most confusing cases, because the most suggestive of gastric disease, are those in which the hepatic flexure and first part of the transverse colon are the seats of the disease; and this may arise not so much from the actual disease in the colon itself, but from the fact that the growth may involve the duodenum. An obstructive effect upon the duodenum soon shows itself by causing very pronounced gastric symptoms. Indeed, so suggestive of pyloric obstruction may these symptoms become that they entirely mask any that otherwise might have led to a suspicion that the colon was implicated. I cannot do better than

illustrate this by the following case: A. B—, æt. 47 years, was admitted to the Victoria Infirmary complaining of stomach disorder. Up to eighteen months ago he had enjoyed good health, but at this time he began to suffer from pain in his abdomen. The pains were always worse about bed-time, lasting for two or three hours. He was troubled with flatulence, but the passage of gas always gave him relief. He also said that vomiting relieved him, and that he would sometimes induce this by putting his finger down his throat. Six months after the commencement of his illness vomiting became a more troublesome symptom, occurring usually at night, and after the pain referred to above. It was usually preceded by nausea. He said that the vomited material was sometimes green and bad smelling, and that occasionally the vomit contained material which had been taken days before. As the vomiting seemed to relieve him, his doctor treated him for some time by lavage, rectal feeding, and a selected and restricted diet. These measures appeared to greatly check the attacks of pain and vomiting. He suffered from constipation, for which he had to take aperients. He had lost four stone in weight during the eighteen months of his illness. External examination of the abdomen revealed no physical signs, and a test breakfast showed the presence of free hydrochloric. When the abdomen was opened a tumour mass was found at the hepatic flexure of the colon, involving the duodenum, the head of the pancreas, and other tissues in the neighbourhood.

The more remote the seat of carcinoma from the cæcum, or the nearer it is to the anus, the more striking are the bowel symptoms; and when a patient, in addition to the gastric disturbances, casually observes that his bowels are irregular, sometimes constipated, sometimes loose, that he has noticed the presence of slime, and perhaps blood also, the fact that the bowel and not the stomach is at fault becomes practically a certainty. It may, in conclusion, be said that when a patient over fifty years of age complains of the insiduous onset and progress of painless dyspepsia, symptoms not adequately explained by a careful investigation of the stomach, the possibility of carcinoma of the colon should always be taken into consideration.

Another disease, not infrequently mistaken for a gastric derangement, is—

Gall-stones.

Here, as in the preceding case of carcinoma of the colon, there are often temporary attacks of so-called "biliousness" manifested by symptoms of dyspepsia, coupled with vomiting. But there is this marked distinction between cancer of the bowel and gall-stones, that while the one is devoid of epigastric pain, the other is frequently associated with it. Possibly one of the most misleading features is the not infrequent occurrence of pain in, or in the neighbourhood of, the epigastrium shortly after the ingestion of food. This arises from the induced functional activity of the biliary apparatus, so that stones in the gall-bladder or cystic duct are at that period liable to produce their disturbing influences. It must always be remembered that jaundice is no necessary concomitant of the presence of gall-stones, and therefore its absence has but little bearing upon the question of differential diagnosis, although its marked existence may be taken as evidence of calculus, or, more correctly, cholangitis. The two symptoms which may be said to possess most significance in distinguishing between dyspepsia the result of gastric disease and that coincidental with gall-stones or other hepatic derangement is the seat of the pain and its character. Thus, with the exception of those cases where there is pyloric ulceration, or ulcer in the first part of the duodenum which would be distinguishable by other symptoms, the maximum seat of the pain invariably exists somewhat to the right of the middle line in the subcostal region, and when of any severity may radiate through to the back up to the right shoulder, and towards the middle line in front. Further, the point, of most marked tenderness, when the abdomen is palpated, is in the right subcostal region. This tenderness is not the simple result of the presence of gall-stones, for they may be present and there be no tenderness, but to the existence of some local peritonitis set up, it may be, but not necessarily, by the existence of stones either within the gall-bladder or within the cystic duct. As regards the character of the pain this is often very variable, and not so distinctive as its situation. When, however, of the conventional colicky type it is much more likely to be due to gall-stones than to any gastric disease, except when the latter is of an advanced nature. Under these latter conditions, however, there will be other

symptoms sufficient to prevent any error in diagnosis. Another factor of some little moment, in distinguishing between gastric derangements dependent upon gall-stones and those due to organic disease of the stomach, is the intermittency of the symptoms in the former case. The dyspepsia or bilious attack may only last for a day or two, and not re-appear for a considerable period, and so far as can be ascertained, is unattributable to any indiscretion in diet which might otherwise suggest it being possibly of gastric origin.

It will not be out of place in this connection to briefly refer to the effect of circulatory changes as causing gastric disturbance. The commonest of these is exercised through obstructive influences upon the portal system. Indigestion, in some form or other, is often a marked feature of—

Hepatic Cirrhosis,

and an abnormally sensitive stomach may manifest symptoms before other marked and significant indications of the disease show themselves.

While obstruction to the return of blood from the stomach may cause gastric derangements, equally may an insufficient supply of the same produce very marked disturbances. Thus—

Arterio-sclerosis

affecting the arteries supplying the stomach is known not only to cause dyspepsia, but to be a fruitful source of epigastric pain. The condition will be diagnosed by the presence of increased blood-pressure as manifested by cardiac signs, and a pulse indicative of high tension and rigidity.

Disease of the Pancreas.

In certain of their phases diseases of the pancreas are very liable at some stage to simulate derangements of the stomach. The anatomical position of the viscus is to some extent responsible for the occasional confusion of the symptoms. Our increase in the knowledge, however, of diseases of the pancreas has, at least, warned us against the possibilities of such a pitfall, and we are now frequently led to a systematic examination of a viscus which we had hitherto too often neglected. When the pancreas is inflamed from any cause, pain and tenderness are experienced in the epigastric region, and therefore in a situation which equally applies to the stomach. So far as the symptom of pain is concerned but little help is derived in differentiating disease of the stomach and disease of the pancreas. It is in certain other symptoms that the distinction must be sought for. Inflammation of the pancreas either in the form of an acute, subacute or chronic affection, and the formation of cysts or other growths, will probably interfere with the normal secretion of the gland, and as the result of certain defective digestive processes fat may be found in the stools. Further, certain changes may be found in the urine—the presence of sugar or reactions of a particular kind elicited by the use of Cammidge's test. Again, jaundice is an occasional symptom due to the common bile-duct being involved in the pancreatic disease. Cysts of the pancreas by upward pressure upon the stomach may cause very marked gastric disturbance, and from their situation lead to the suspicion that the growth is connected with that viscus. In some instances it will only be by an exploratory operation that the true nature of the disease will be determined.

The stomach is liable to be very seriously deranged by a—

Movable or Floating Kidney.

There may, at times, be very obvious indications of dilatation, coupled with flatulence and other dyspeptic symptoms, which may naturally suggest pyloric obstruction as a possible cause. It will not, however, be difficult to exclude this by a closer investigation into the gastric condition; and any doubt will at once be cleared up in favour of a renal cause by the detection of a dislocated organ in the right loin.

Very prolific sources for the production of gastric derangement are to be found in certain diseases of the—

Nervous System;

and from the point of view of diagnosis they are frequently among the most misleading. Under the vague and somewhat inexplicable systemic malady known as "neurasthenia," we have a class of sufferers whose predominating manifestation is often some form of stomach trouble. So preponderating may the symptoms referable to the stomach be that they frequently lead to the belief that, whatever other symptoms are present, they are probably and primarily attributable to some organic

mischief directly connected with that viscus. In some of the more advanced cases actual disease does exist, and a chronic gastric catarrh may prove to be a very evident complaint. However, a careful investigation into the whole circumstances surrounding the case will usually lead to a correct diagnosis. It may, for instance, be found that a very strong vein of neurosis runs through the history of the case, not only in relation to the patients themselves, but often also in association with their families. Again, other symptoms will exist suggestive of systemic debility of a nature not to be explained solely on the basis of a local gastric trouble. It may be incidentally remarked here, in order to emphasise the difficulties which this class of case may occasionally present, that instances have occurred, not only in my own hands, but in that of other surgeons, where the extreme preponderance of gastric symptoms has led to an exploratory operation. Nothing of an organic nature has been discovered, and nothing more than the exploratory operation has been performed, but the result has been good. The powerful influences exercised upon the patient, however they are to be explained, seem to have had a beneficial effect upon the neurotic condition. It must, however, be said that anything like indiscriminate operation in this class of case is bad; and will, if practised without due regard to other considerations, bring discredit to the operation, and sometimes harm to the patient.

As regards actual organic diseases of the brain and spinal cord, we frequently have gastric symptoms, which, in the initial stages, are only too likely to mislead. In the case of the brain, it may be a tubercular, or other kind of tumour; or it may be tubercular meningitis. The chief gastric symptom is usually vomiting. But the involuntary and almost spasmodic ejection of the contents of the stomach, unprovoked apparently by any errors of diet, and devoid of other evidences of gastric derangement, is sufficient to awaken suspicion, and to direct attention to the possible existence of disease elsewhere than in any part of the alimentary canal. With regard to the spinal cord and its membranes, such diseases as locomotor ataxia and caries of the spine are apt to produce epigastric pain, which has not infrequently proved misleading. With children, more particularly, the epigastric pain associated with caries in the region of the sixth dorsal vertebra is often attributed to the stomach rather than to the spine. The rarety, however, with which children suffer from more or less constant gastric trouble should always lead to a careful examination of the spine. In locomotor ataxia the pains have the characteristic lightning-like peculiarity; and while other indications of gastric derangement may be absent there is usually the co-existence of similar "shooting" pains in other parts of the body. The mistake of wrongly attributing the epigastric pain to gastric disease is rarely made when the case is carefully gone into. Nevertheless, errors of treatment have occurred, and the stomach has been explored by operation before the true nature of the complaint has been diagnosed.

It would be easy to add to those complaints which have already been described others which produce or simulate gastric derangements; but I cannot do more than enumerate some of these; and so long as the diagnostician bears them in mind he will not be likely to overlook the true source of the symptoms. Diseases such as angina pectoris, pericarditis, acute pneumonia and pleurisy mislead by the production of pain in and about the epigastric and umbilical regions. On the other hand, aneurysm of the aorta and of some of its main branches, tumours of the omentum and mesentery, may by direct pressure upon the stomach cause other symptoms than those of a simple painful nature. Anæmia, from whatever cause, renders the stomach—as it does, indeed, all other organs—peculiarly susceptible to derangements. The gastric symptoms may sometimes be so pronounced as to overshadow other signs in themselves more distinctive of the disease upon which the former solely depend.

January 25th, 1909.

Neuralgia. — Reyburn holds that chronic neuralgia is due to a purely physical cause, viz., increased blood-pressure. In addition to the usual remedies he has given ergot in full doses, and, on theoretical grounds, with excellent results. It is especially useful in the chronic neuralgia of over-fed women in the higher walks of life, who take little or no exercise. A fluid drachm three times a day, with forced exercise and diminished food supply, is very efficient.—*Journ. A. M. A.*, vol. lii, No. 1.

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THE TREATMENT OF SYPHILIS BY ARYLARSONATES.*

By J. ERNEST LANE, F.R.C.S.,
Surgeon and Lecturer on Surgery, St. Mary's Hospital, Senior Surgeon, London Lock Hospital.

ANY advance in the treatment of syphilis is a step forward in the march of medical science, and any efficient addition to the therapeutic measures at our disposal will be heartily welcomed by the medical profession. We have hitherto been compelled to rely solely upon one drug, viz. mercury, as an antagonist to the virus of syphilis, though it may be granted that the preparations of iodine are of value in subduing certain of the symptoms and products of the disease; but although mercury is hardly likely ever to be degraded from its present rank as a therapeutic of this most formidable malady, yet it is not without its disadvantages and its drawbacks, and there is ample scope for any remedy which might rival it or even compete with it in its effect upon syphilis. Our treatment of syphilis in the immediate past has been somewhat empirical in its character, and it was a lucky stroke that the drug selected for its treatment in the remote ages was one which is now proved to be antagonistic to the micro-organism of the disease, and one which is in accord with modern ideas of its pathology. The treatment of syphilis with mercury dates back to at least the fifteenth century, and although it has on several occasions since that era met with disparagement and even discredit, yet no efficient rival or substitute has ever been introduced. Certainly arsenic *has* been utilised in the treatment for half a century or more, either alone or in combination with mercury, in the form of Donovan's solution, but it is only since the discovery of the micro-organism of syphilis by

* Delivered before the Medical Graduates' College and Polyclinic.

Schaudinn in 1905 that its merits have been sufficiently recognised. Since that date the treatment by arsenical compounds has met with many advocates, and it has been thoroughly established that intra-muscular injections of arsenical preparations are not fraught with the dangers and disadvantages which are inseparable from its administration by the mouth. In this respect there is a great resemblance between mercury and arsenic, for there is little doubt that the value of the former drug has been greatly enhanced by this method of intra-muscular injections, the merit of which is now universally acknowledged. Intra-muscular injections of arsenic, in the form of atoxyl were recommended for the treatment of sleeping-sickness by Koch, who found that the prolonged administration of this drug rendered a patient suffering from trypanosomiasis free from trypanosomes, and so susceptible of a cure of his otherwise almost certainly fatal disease. Atoxyl is one of the arylarsonates, *i.e.* organic salts, containing arsenic in combination with an aromatic radicle; it is the mono-sodium salt of para-amino-phenyl-arsenic acid, and contains 29·10 per cent. of arsenic. Administered in this form intra-muscularly it enables the surgeon to introduce comparatively large quantities of the drug without any toxic effects, and thereby to obtain the full therapeutic effect without any of its disadvantages. The arylarsonates should not be given by the mouth, as the action of the gastric secretion causes them to break up, and to give rise to symptoms of arsenical poisoning. The treatment by atoxyl was found to be equally successful in other forms of protozoal infection, such as in malaria, in pellagra, in the tsetse-fly disease in horses and cattle, in the dourine disease of horses (a condition in some respects resembling syphilis, and transmitted by coitus, and consequently known as the "mal-de-coit"), and in the spirillosis of hens. Since the action of atoxyl on diseases due to protozoal infection had been clearly demonstrated, and since the *Spirochæte pallida* was presumably protozoal in nature, there was a natural inducement to try the effect of atoxyl on this newly discovered protozoon, and numerous observations have been recorded as to its effects upon the syphilitic virus. The late Professor Lassar first introduced the treatment by intra-muscular injections of arsenical preparations in 1902, but abandoned it for a time; on resuming the treatment with greatly increased doses he met with more satisfactory results. In France atoxyl has been strenuously advocated by Hallopeau, who, in an experience based on 120 cases, found that it exerted a marked therapeutic action on syphilitic processes, especially in the primary and early secondary manifestations of the disease; in the later secondary and tertiary manifestations the results of this treatment were not found to be equally satisfactory. It must be confessed, however, that though Hallopeau has brought forward such a formidable list of cases treated by atoxyl, for the most part with satisfactory results, he does not seem to have been able to convince the majority of his French colleagues of its superiority over other methods. Indeed, most of the early advocates of the atoxyl treatment found that there were counter-balancing disadvantages, and that intolerance of the drug often early manifested itself; this was characterised by gastro-intestinal disturbance, vomiting, nausea, headache, giddiness, fever, frequent micturition, retention of urine, and last, but not least, by temporary or permanent blindness. This is a somewhat formidable list of the possible sequelæ of this treatment, and is in itself quite sufficient to cause anyone to pause before he submitted his patient to risks of such grave disaster; unless some explanation of these possible contingencies had been forthcoming, the treatment would ere now long have been abandoned. But in almost every remedy there is a risk, which is usually proportionate to the effects of such remedy, and unless the risks are counter-balanced by the benefits which accrue, the remedy is inappropriate and must be relegated to the category of therapeutic failures—by this time a not inconsiderable one. Further researches, however, appeared to prove that the preparations of atoxyl used were unequal as regards their effect upon the individual patient; that in some cases its action was cumulative and toxic symptoms early supervened, whilst in others, though the drug was readily tolerated, it exercised no marked effect upon the symptoms. Efforts have recently been made in this as in other countries to devise some arsenical preparation of stable composition and low toxicity, possessing all the therapeutic virtues of atoxyl, and devoid of its many vicious propensities already enumerated. In Germany, Ehrlich has introduced a modification of atoxyl in the

form of arsacetin, a sodium acetyl-phenylarsonate, which has been recommended by so eminent an authority as Professor Neisser, who states that it has far less toxic properties than atoxyl, that it has a far better therapeutic effect upon syphilis than that preparation, that its efficacy is not prejudiced by keeping it for a long time in solution, and that it is not affected by even a daily boiling of the solution. Neisser has proved that syphilis in animals can be aborted by the early administration of arsacetin, and further, that it will clear up the symptoms of constitutional syphilis; and he has applied the knowledge acquired from the experimental syphilis of animals to the human subject; he recommends the administration of arsacetin and mercury simultaneously, and states that the combined action of those drugs has given good results. His method of administering arsacetin was by two injections a week on two successive days of nine grains of the preparation, until twenty injections had been given.

For much of our recent advance in the treatment of syphilis in this country we are indebted to the labours of the advisory board of the officers of the R.A.M.C. who have taken full advantage of the unusual opportunities afforded them for the study and treatment of venereal diseases. In this field the name of Col. Lambkin stands out conspicuously as one of the pioneers of the treatment of syphilis by intra-muscular injections of mercury. But he has not confined his attention to that method of treatment to the exclusion of all others, and has recently published his experience of the treatment by the arylarsonates. He commenced by employing atoxyl in the dosage recommended by Hallopeau, 11½ gr. being first given, two days later 10 gr., and three days later 7¾ gr., a similar course being administered in ten days' time; his first series of cases numbered nineteen, and the results of the treatment were described as encouraging. It was found, however, that as the demand for this substance increased its quality deteriorated, and many cases of toxic poisoning from it were recorded. Lambkin in consequence abandoned the use of atoxyl, and in his next two batches of cases, amounting to fifty-five, utilised a preparation called "soamin" which was manufactured by Burroughs & Wellcome, and which contains 22·8 per cent. of arsenic. He gradually increased the dosage of this preparation, and administered it in doses of 10 gr. every other day until 100 gr. had been taken. In his opinion, contrary to that of Neisser, the arylarsonates should not be administered simultaneously with mercury, as the latter drug dissociates the ingredients of the former and so may give rise to toxic symptoms. Lambkin concluded that soamin given early and in sufficient quantities was a prophylactic against any further development of the disease; that it delayed and modified the secondary symptoms; that it had a markedly beneficial effect on all syphilitic ulcerations, and that it might be considered as a second specific for syphilis. A further series of cases amounting to thirty-four has recently been brought forward by the same writer, twenty-six having been treated by soamin and eight by arsacetin, the latter preparation being administered in doses of 40 minims of a 15 per cent. solution, *i.e.* 7 gr. of arsacetin every second day, ten injections constituting a sufficient course. Lambkin could not at present state positively that the arylarsonates were able permanently to destroy the *Spirochæte pallida* and so effect the cure of the disease, but ten of his cases had been under close observation for the past ten months, and had developed no further symptoms of syphilis.

The treatment by "soamin" has also been recommended by Mr. Pugin Meldon, surgeon to the Westmoreland Lock Hospital, and he is of opinion that it is a very valuable addition to the number of anti-syphilitic remedies at our disposal. He advises its administration in those who cannot tolerate mercury in sufficient quantity to relieve their symptoms, especially those cases in which extensive ulceration of the throat and mouth is present; in cases of syphilis complicated with tuberculosis, also in patients who will not undergo a long course of mercurial treatment. A few weeks of soamin injections gave a far better chance of warding off further development of the disease than an insufficient treatment by mercury.

In comparison with Col. Lambkin's, my experience of this treatment has been limited to such a short period of time and to such a small number of cases that I have had grave misgivings as to whether I was justified in making a communication on this subject. The cases I am about to bring forward are in themselves obviously insufficient material on which to base any conclusion, and further, the short time they have been under obser-

vation would render them absolutely valueless were it not for the fact that they are confirmatory of the opinions expressed by Lambkin and Hallopeau, who have had a far wider experience of the treatment than I have. Though I commenced this treatment in July, 1907, *i.e.* before Col. Lambkin initiated it in this country, I met with early disappointment and discontinued it very shortly; but my failure at the commencement was in no sense to be attributed to the shortcomings of the drug, but to the insufficient dosage I then employed. In July and August, 1907, I treated four cases of syphilis with intra-muscular injections of atoxyl, the dosage being $7\frac{1}{2}$ minims of a 10 per cent. solution, an amount which, according to knowledge acquired since that time, was obviously insufficient. From the point of view of any communication as to the efficacy of aryl-arsonates these four cases are not worthy of one moment's consideration; I only allude to them to show that I was early alive to the possible value of this form of treatment.

In the present year I have resumed the treatment, but have utilised a different preparation to any of those previously mentioned, a substance called "orsudan," which, like soamin, is a production of Burroughs & Wellcome. The formula of this compound is sodium, 3 methyl, 4 acetyl-amino phenylarsonate, so the abbreviation of its name is perfectly justified. It is anhydrous and contains 25·4 per cent. of arsenic—nearly 3 per cent. more than soamin—is soluble in three times its weight in water, and is less toxic than soamin. This preparation has been used by me on twelve cases, some of them presenting only the primary sore, but others with obvious secondary manifestations. It was administered in doses of 10 gr. dissolved in half a drachm of distilled water heated up to 100° F. The injections were introduced, on alternate days, deeply into the muscles of the buttock, in the situation I have indicated on a previous occasion as being free from any large vessels or nerves; they produced very little pain, and were in this respect distinctly superior to calomel injections, and in no case was any lameness or pain on locomotion experienced later. In two cases the interval between the injections had to be slightly lengthened owing to the supervention of diarrhœa, and in one case vomiting followed upon the first injection, though it never occurred subsequently. None of the grave symptoms of arsenical intolerance already alluded to were observed, and in no case did any disturbance of vision supervene. As regards the results, they are, in my opinion, most satisfactory, and such as could not have been obtained by any form of mercurial treatment, not even by intra-muscular injections of calomel, which I have hitherto regarded as the most efficient treatment of syphilis, and the one which caused the most rapid disappearance of symptoms, and also the one from which relapses were least frequently encountered. As to how long this treatment will guarantee an immunity from subsequent manifestations of the disease I am not at present in a position to speak, but judging from the experience of Col. Lambkin, to which I have alluded, there is hope that, in a large proportion of cases, the immunity may be permanent. On this point I hope to have something to say on some future occasion, for I certainly intend to follow up the treatment systematically in the future unless some counter-balancing disadvantages present themselves, a contingency which at present I do not anticipate. In comparison with Col. Lambkin and his coadjutors in the field of syphilography in the R.A.M.C., I labour under considerable disadvantage, for, with the class of patients who present themselves for treatment at the London Lock Hospital, it is almost impossible to impress upon them the importance of their being kept under periodical supervision; consequently, when they leave the hospital, free from all symptoms of disease, they neglect to report themselves, and are, for the most part, completely lost to view. In the Army Medical Department, on the other hand, the subjects of the disease are under constant discipline and supervision, and any relapse or subsequent developments of the disease will at once come under notice and treatment.

It may, of course, be urged that in many of the cases treated on this plan an incorrect diagnosis has been arrived at, since the treatment has been commenced before the appearance of secondary symptoms, for undoubtedly in many instances it is a matter of difficulty, and even of impossibility, to make a diagnosis of syphilis from the appearance and feel of the sore; but the cases I selected were typical ones, and ones in which I should have confidently awaited the supervention of unequivocal signs of the disease in the form of some secondary syphilitic manifestation. But even in cases in which

there is an element of doubt as to the nature of the initial manifestation, it would surely be better to give this method the preference over any form of mercurial treatment, since, according to our present knowledge, only three weeks is required for the treatment by arylarsonates in comparison with the far longer period which is necessitated for any efficient course of mercurial administration. Another powerful argument in favour of this treatment, especially if the disease is encountered in its primary stage, is that by its agency the customary secondary symptoms appear to be completely abolished; now, it is by these secondary lesions that the disease is to a great extent propagated, mucous tubercles about the genitals or on the buccal mucous membrane being a most fruitful source of contagion, for the patients are so frequently ignorant of their significance and danger. If, then, these sources of contagion are abolished, the spread of the disease must be to some extent modified, so that from the point of view of prophylaxis the treatment is a valuable one.

My list of cases which have been treated by orsudan amounts at present to only twelve, of which cases I give here a brief outline. In three of these cases, Nos. 2, 4 and 8, the treatment was commenced before the appearance of any secondary symptoms, and they all left the hospital free from any signs of disease, and after the lapse of a sufficient period for syphilitic manifestations to have shown themselves in the ordinary course of events. In the remainder of the cases secondary manifestations were present, and so in these the diagnosis of syphilis cannot be disputed. All these patients left the hospital free from all symptoms of syphilis, but the last one had still some mucous patches on his tonsils after the completion of his ten injections. In all the cases, with two exceptions, the full course of 100 gr. was administered, as doubtless would have been the case with these other two patients, had not one absconded after the ninth injection, while the other had to be dismissed from the hospital, after having had six injections, for fighting.

As to the method in which these arsenical compounds act, it is impossible to say whether arsenic has a destructive effect upon the *Spirochæte pallida* as mercury is supposed to have, or whether it acts by stimulating the phagocytes to a more strenuous resistance of the protozoal invasion, and this is the view which is mostly held as to the action of the arylarsonates. If mercury acts in the same way, as a stimulant to phagocytosis, its action in this direction is not a very rapid one, for the spirochæte is found in large quantities in the syphilitic lesions of those who are under mercurial treatment, even in the stage of tertiary symptoms. It seems to me that the action of mercury and of arsenic upon syphilitic manifestations is totally dissimilar, for the arylarsonates appear to have a much more rapidly destructive effect upon the germ to which the disease has been proved to be due; according to Ringer arsenious acid is poisonous to many of the lower forms of animal life, and from the action of the arylarsonates on syphilis it appears to me to be quite reasonable to suppose that they have a destructive action on the *Spirochæte pallida*.

Notes of Twelve Cases Treated by "Orsudan."

Case 1.—A. B—, æt. 22 years; admitted September 14th, 1908, with enormously swollen and œdematous penis, and a general desquamating papular eruption. Had ten injections of orsudan on alternate days, the first one of gr. v, the others of gr. x. He was discharged on November 3rd, free from symptoms, and was instructed to report himself, which he has been doing until recently.

Case 2.—N. S—, æt. 20 years; admitted September 22nd, with an indurated sore of three weeks' standing, the sore being of cartilaginous consistence. No further signs of syphilis. Given ten injections of orsudan gr. x. Dismissed October 13th, free from symptoms; last seen November 11th, nearly six weeks after admission and nine weeks after appearance of sore, but still no further sign.

Case 3.—C. D—, æt. 25 years; admitted September 29th; had had concealed sores and phimosis for the past two and a half months, for which he had had two weeks' mercurial treatment. On admission, he had enormous swelling of prepuce and phimosis, with inguinal adenopathy and ulceration of fauces. He had ten injections of orsudan gr. x on alternate days, with an intermission of six days after the ninth injection on account of diarrhœa. Left hospital on November 30th, *i. e.* after stay of two months, with absolutely no trace of disease.

Case 4.—E. F—, æt. 22 years; admitted

October 6th; had phimosis for seven weeks, and when admitted had an indurated mass perceptible under prepuce, and enlarged and indurated glands in left groin; was given ten injections of orsudan gr. x at intervals of one day, with one interval of four days towards the end, *i. e.* after the ninth injection, owing to diarrhœa; still under observation and in hospital, but showing no symptoms. *N.B.*—On subsidence of phimosis in this patient, there was a typical indurated sore on inner surface of prepuce.

Case 5.—G. H—, æt. 20 years; admitted October 7th; had been an out-patient at another hospital seven weeks previously, when he was told he had syphilis and was put on treatment for that disease. On admission to the Lock Hospital, he had an indurated sore round corona, a faint macular syphilide, condylomata ani, and ulcerated throat. Given ten injections of orsudan gr. x; no symptoms after seventh injection. Discharged on November 4th, free from symptoms, but to remain under supervision.

Case 6.—I. J—, æt. 22 years; admitted October 17th. Had superficial sores on prepuce and phimosis for five weeks; enlargement of left inguinal glands and macular eruption of one day's standing. Given ten injections of orsudan, gr. x. Had prepuce slit up on October 24th, disclosing indurated sore which had healed on October 31st. No further symptoms; discharged on November 6th free from all signs of syphilis, and recommended to remain under observation.

Case 7.—K. L—, æt. 20 years; admitted October 17th. Noticed sore one month ago, followed by rash two weeks later. Had phimosis and discharge from under prepuce, also a macular syphilide on face and abdomen. Had nine injections of orsudan gr. x. Prepuce slit up on October 25th; no further signs of disease. Went away November 4th, before completion of course, but free from symptoms.

Case 8.—M.N—, æt. 23 years; admitted October 27th. Had sore under prepuce for three weeks and phimosis one fortnight; circumcised October 30th, when indurated sore of corona was exposed; inguinal adenopathy both groins. Given ten injections of orsudan, gr. x; vomited on morning after first injection. Discharged on November 18th, free from symptoms, but instructed to report himself at intervals.

Case 9.—O. P—, æt. 22 years; admitted November 2nd. Had sore under prepuce for five weeks and phimosis for a fortnight. Hæmorrhage from under prepuce for four days; penis swollen and œdematous, and suppurating gland in left groin; slight macular rash on front of abdomen and chest. Circumcised on November 6th; phagedænic ulceration disclosed, which had destroyed most of glans penis, and had extended into urethra, so that urethral fistula was present posteriorly. Had pustular eruption on back and ulceration of fauces. Given ten injections of orsudan, gr. x; on December 12th ulceration of penis almost healed and rash had almost entirely disappeared.

Case 10.—R. S—, æt. 30 years; admitted November 9th. History of sore twelve weeks previously, followed by extensive phagedæna on undersurface of penis, opening into urethra in three or four places. Had six injections of orsudan, gr. x, at end of which treatment all ulceration was healed, but patient was dismissed on November 22nd for fighting.

Case 11.—T. U—, æt. 48 years; admitted November 14th. Had a sore for six weeks and phimosis for four weeks; treated for syphilis at general hospital for a few days at commencement of symptoms; had œdema of penis and sinus on dorsal surface near base of prepuce; indurated sore perceptible on inner surface of prepuce. Given ten injections of orsudan, gr. x; no further signs of syphilis; still under observation.

Case 12.—V. W—, æt. 33 years; admitted November 11th. Has had sore, phimosis, and indurated glands in right groin for four weeks; distinct macular eruption. Had ten injections of orsudan, gr. x, at the end of which treatment the rash had almost disappeared, but he had then some mucous patches on tonsils; still under observation.

February 1st, 1909.

Typhoid Pyonephrosis.—Meyer and Ahreiner report a case in a girl, æt. 16 years, a chronic bacillus carrier. She had passed through measles, scarlet fever, whooping-cough, pneumonia, typhoid with left pyonephrosis and diphtheria, all before the age of nine. Trouble recurred in the left kidney, and it was removed after eight months of symptoms. The typhoid bacilli were voided exclusively in the urine, which became sterile after the nephrectomy.—*Journ. A. M. A.*, January 2nd.

A CLINICAL LECTURE
ON
MUSCULAR RHEUMATISM.

By WILLIAM MURRELL, M.D., F.R.C.P.,
Physician to Westminster Hospital and Lecturer on Medicine and Clinical Medicine.

MUSCULAR rheumatism is a chronic painful affection of the white fibrous tissue connected with the muscles, and implicates the aponeuroses, tendons, fibrous septa, fasciæ and adjacent periosteum. It is an inflammation and hyperplasia of the connective tissues occurring irregularly and in patches, so that it gives rise to the formation of swellings, nodules and indurations of various sizes. These thickenings when superficial are easily detected on palpation; they are tender and are the seat of a continuous dull, aching pain. The joints themselves are not implicated and the disease is not merely a chronic form of acute rheumatism, although, in many instances, it may be microbic in origin. It is often spoken of as a myalgia, but the two conditions are not identical. In cases of omodynia or pain in the shoulders it is often difficult to determine whether the pain lies in the articulation or in the overlying structures. When muscular rheumatism is accompanied by slight febrile disturbance the condition is often described as an "influenza cold," but there are none of the characteristics of the more serious disease and it is not followed by the sequelæ of that affection.

The attack usually comes on suddenly as the result of exposure to wet or cold, or possibly from standing or sitting in a draught. It commonly attacks middle-aged people, preferably those with a gouty tendency. There is little or no constitutional disturbance, and there is little, if any, elevation of temperature. The last point is illustrated by a severe case of muscular rheumatism recently under my care. The patient, a carpenter, æt. 34 years, living at Eastbourne, was attacked suddenly with pain and stiffness in the muscles of the left thigh and in the right biceps and lumbar region. He stated that for some days previously he had been working in an open shop exposed to a cold east wind. He sought relief, not because he felt ill, but from inability to follow his occupation. His temperature was taken systematically for ten days and only twice rose to 100° F.

With regard to the pain, as a rule, though not universally, one group of muscles or a group of muscles functionally related is attacked. This is so generally recognised that the nomenclature and classification are typographical. The pain, however, may be widely distributed and is always increased by movement, so that the patient has perforce to lie still and is glad to remain in bed. The attack may last only a few hours, but usually persists for days or even weeks, the duration depending to some extent on the mode of treatment adopted.

Many cases of so-called muscular rheumatism are in reality examples of myalgia. A familiar form is the stiffness which follows the first day's hard ride after a long period of enforced idleness and inactivity. A similar condition is met with in young women after a bout of dancing for which they are not in training. Delicate middle-class women who have to dispense with a servant and perform domestic duties suffer severely in this way when debilitated by child-bearing, suckling, or profuse menstruation. Another example is the "cutting-out pain," the result of an unusual strain on the back and loins in leaning over a table to cut out patterns. A sub-variety of this is the "trying-on pain." against which fashionable dressmakers and tailors guard their customers by the injudicious administration of a glass of sherry or a whisky-and-soda. A similar condition is met with in the girl pupils of old-fashioned schools, who are compelled to perform pianoforte exercises in a cold, damp room before breakfast. A more pleasing variety is the pain in the intercostal muscles, the "laughing until the sides ache" which is experienced at an amateur theatrical performance or the first night of a society play. Myalgia of the pectoral muscles follows a severe attack of coughing in cases of phthisis and of chronic bronchitis with emphysema. A similar condition affecting the muscles of the abdomen is seen in the persistent vomiting of sea-sickness and pregnancy. The artificial support afforded by stays will enable a woman to sit up long after her male companions have assumed a reclining position in an arm-chair. The stiffness of pedestrians and cyclists and the cramp of swimmers are true myalgias, although they are often spoken of as rheumatism. In all

these conditions the muscles are called on to perform, without adequate training, functions beyond their strength. The favourite seats of muscular rheumatism are the neck, chest and loins, and special names are given to the complaint according to the particular group of muscles implicated.

(1) LUMBAGO is a painful affection of the muscles of the loins and of their tendinous attachments, a condition not infrequently associated with sciatica or sciatica neuritis. It probably arises in the termination of the sensory nerves between the muscular fibres, in what are called the "muscle spindles." The extension of the pain from the substance of the muscle to the tendinous structures may be regarded as a justification for locating the site of the affection in the fibrous tissues in which the sensory nerve-endings are situated. The attack usually comes on suddenly, and the sufferer finds that he is unable to stoop down or more commonly to regain the upright position. This condition has to be differentiated from the onset of certain acute specific diseases, such as smallpox and influenza, from lumbar caries and aneurysm, from uterine mischief, from malignant disease of the rectum, from the passage of a renal calculus, and sometimes from appendicitis. In some cases the pain may be traced to disease of the articulations of the lumbar vertebræ. In the lay mind backache is supposed to indicate the existence of kidney mischief, but as a matter of fact Bright's disease is rarely attended with pain in this region.

One of the most speedy methods of relieving the pain of lumbago is acupuncture, the needle being inserted for a couple of inches with antiseptic precautions on both sides over the seat of the greatest pain or tenderness. Ether spray or freezing with chloride of ethyl is another valuable mode of treatment which affords relief even in cases of long-standing. Poultices, turpentine fomentations, and the application of an ice-bag have their advocates, whilst electricity of sorts is not without its adherents. A liniment of aconite, belladonna, chloroform and capsicum is a favourite formula as a local application. A useful remedy in these cases is amber oil (oleum succini), obtained by the distillation of amber. It is described as a stimulant, anti-spasmodic, and counter-irritant. I am not prepared to say that it is more efficacious than other members of the same class, but it has yielded good results in my hands, and I have used it fairly constantly for twenty years. About half a drachm should be rubbed in night and morning. Rubbing of all kinds, whether called massage or by some more homely name, is useful in all forms of muscular pain. It is by no means a modern method of treatment, and in 1825 John Grosvenor, of Oxford, published a book in which he described a system of friction for the cure of rheumatism and other allied affections. The application of bandages applied firmly to the affected parts often affords speedy relief, but again it is by no means a new method of treatment, for Dr. William Balfour, in 1816, published a work on a method of curing rheumatism, in which compression by the use of bandages is strongly recommended. The thermic hammer and the Paquelin cautery may be tried in obstinate cases. Salicylate of sodium may be used when there is a rheumatic tendency, and in certain conditions, especially when the pain is worse at night, the iodides are indicated. The compound tincture of guaiacum has its adherents, and the old-fashioned formula known as "Chelsea Pensioner" is said, in obstinate cases, to have worked wonders. In acute cases a hypodermic injection of morphine over the seat of pain may be resorted to, although it is not a popular mode of treatment with busy practitioners.

In connection with lumbago and other forms of muscular rheumatism, reference must be made to the indurations or thickenings of the fibrous tissues, to which attention has been called by Stockman, of Glasgow. They are now regarded as an essential constituent of the disease. They are well defined and circumscribed, varying in size from a small shot or split pea to an almond or even half a walnut. Sometimes they are deeply placed, and at others are quite superficial in the subcutaneous tissue. When pressed upon they give rise to pain, especially when the branch of a nerve is involved. One such nodule in the lumbar region may be enough to give rise to persistent lumbago. They consist of fibrous tissue, and an amorphous sero-fibrinous matrix; fibroblasts are not numerous, and there is no congregation of leucocytes in the neighbourhood of the inflamed area. The swelling is a chronic, but comparatively localised inflammation of the perimysium. They never undergo osseous change or infiltration with urate of sodium,

nor do they suppurate. These points are of diagnostic importance.

These nodules are closely allied to the subcutaneous nodules described by Barlow and Warner (1881) as occurring in children the subjects of rheumatism and chorea. In these cases the back of the elbow, the malleoli and the margins of the patella are the commonest sites. Other situations are the neighbourhood of the vertebral spines, the spine of the scapula, the crista ilii, the extensor tendons of the foot and hand, the temporal ridge, the forehead, the superior curved line of the occiput and the pinna of the ear. On section they present "wavy strands of fibrous tissue, with caudate spindle-shaped nucleated cells and abundant vessels." These nodules had been previously described by Meynet, Rehn (1878), Hirschsprung of Copenhagen, and in adults by Troisier and Brocq (1881).

Heberden's nodes, described by William Heberden in his 'Commentarii de Morborum' (1802), are quite distinct from, and have nothing in common with, these fibrous thickenings. His "Digitorum Nodi" are osseous structures situated on the lateral aspects of the terminal joints of the fingers. He states that they are more common in women than in men, and are usually symmetrical. They are as a rule painless, but may give rise to a certain amount of discomfort by pressure on adjoining structures. In connection with them are often small cysts which have been described as herniæ of the synovial membranes. Concurrently with the appearance of the nodules the joints of the fingers often become swollen, tender and inflamed. It is impossible to avoid the conclusion that Heberden's nodes are allied to the next form to be described.

John Haygarth, in 1805, published a clinical history of 'Nodosity of the Joints,' of which he had seen thirty-four cases, all, with the exception of one, being in woman. The joints chiefly affected were the fingers. These Haygarth's nodosities were not separate tumours, but felt as if they were enlargements of the bones themselves, from which it may be gathered that they were osseous growths. He says the joints were painful, and often felt sore to the touch. As the disease increased the joints became distorted, and, in inveterate cases, even dislocated. In some cases a crackling noise was perceived in the joints. He lays great stress on the fact that the disease has no intermissions, and but slight remissions. During the remainder of the patient's life the nodes gradually enlarge, impeding more or less the motions of the limbs. The malady spreads to other joints without leaving or producing even an alleviation in those which had been previously attacked. The author points out that the nodosities, in their gradual progress, sadly embitter the comforts of life, but do not shorten its duration. This is obviously a good rough clinical description of what was then beginning to be known as rheumatic gout. Haygarth's nodosities and Heberden's nodes must be regarded as symptoms of what we now call osteo-arthritis.

Gouty nodules, or tophi, can hardly be mistaken for rheumatic nodes. They are due to the deposit of sodium biurate in the tissues of those parts of the body in which the circulation is not very active. They are seen in the most typical form in the helix of the ear, where, however, they attain no great size, the majority not being larger than a pea. Larger masses, some as big as a hen's egg, may occasionally be seen on the dorsal aspect of the forearm, and may be symmetrical. On the knuckles, in the form of what are called chalk stones, they are often accompanied by considerable deformity of the joints.

Parrot's nodes, or bosses, are low elevations on the parietal eminences caused by infantile periostitis, usually the result of syphilis, so that from the point of view of diagnosis they give rise to no difficulty.

(2) PLEURODYNIA may be due to muscular rheumatism, thoracic myalgia, or intercostal neuralgia. It has also to be differentiated from dry pleurisy, an attack of herpes zoster or zona, from fracture of the ribs, possibly as the result of a violent fit of coughing, from syphilitic periostitis and other similar conditions. The diagnosis as a rule presents no difficulty. In intercostal neuralgia there are usually three tender points, corresponding to the posterior, lateral, and anterior branches of the intercostal nerves. In the treatment of pleurodynia, from whatever cause arising, local rest is of importance, and can be ensured by the application of a stout belladonna plaster, perforated for preference, or a broad bandage. Counter-irritation in the form of blisters, or a mustard poultice, will be found useful. When the urine is scanty and loaded with lithates diuretics may be given, and anæmia may be removed by the judicious adminis-

tration of iron. The prognosis is good, and the patient usually recovers in a few days.

(3) RHEUMATIC TORTICOLLIS, or stiff neck, is an affection of the muscles of the anterior lateral region usually induced by exposure to cold. It has to be distinguished from the stiff-neck symptomatic of disease of the upper part of the spine, from rupture of the fibres of the sterno-mastoid, and from meningeal affections. In congenital torticollis, or wry-neck, in which the sterno-mastoid is primarily at fault, facial asymmetry on the affected side is usually observed. The eyes are not on a level, nor are the angles of the mouth, the cheek is wasted, and the nose deviates to one side. When this condition is slight it may sometimes be detected by observing the patient's face in a looking glass. The non-congenital clonic torticollis of children is often of neurasthenic origin, and may be cured by remedies and modes of treatment which appeal largely to the imagination. In some cases the disease is of reflex origin. The spasmodic torticollis of adults, which is a disease of the spinal accessory nerve, and to some extent of the posterior branches of the cervical spinal nerves, belongs to an entirely different category. The muscles affected are the sterno-mastoid, the trapezius, and the spleneus. It may be either tonic or clonic, and in both forms the muscles involved become hypertrophied. There is usually a considerable amount of pain, but there may be only a persistent sense of fatigue. It is a functional neurosis, and in some cases is allied to the craft palsies. Medical treatment is, as a rule, not efficacious, although large doses of the iodides sometimes do good. Mechanical appliances are worse than useless. Nerve-stretching may do good, but neurotomy, or, better still, the removal of a portion of the nerve implicated, offers a greater prospect of success. The purely rheumatic form presents very little difficulty, and may be treated on the lines already laid down for muscular rheumatism, attention being paid to rubbing, electricity, and the use of counter-irritants in the form of stimulating liniments.

There are other conditions, accompanied by pain, which might momentarily be mistaken for muscular rheumatism. The onset of many acute specific diseases is attended with pain; for example, in enteric fever, the onset of which is usually insidious, pain in the limbs is by no means an unusual feature. In some forms of influenza, pain in the loin is a common symptom, and the patient may be under the impression that he is suffering from nothing worse than an attack of lumbago. The pains of malarial fever, of beri-beri, and of dengue or "break-bone" fever, are well known.

Some cases of so-called muscular rheumatism are due to localised neuritis, which may be alcoholic in origin, but are more frequently the result of an old and imperfectly treated attack of syphilis.

February 1st, 1909.

NOTES FROM DR. RUDOLPH SMITH, STOCKTON-ON-TEES, COMPLETING THE CASE OF ACUTE TUBERCULOUS PERITONITIS IN A HOSPITAL NURSE, TREATED BY TUBERCULIN GIVEN BY MOUTH, DESCRIBED IN OUR ISSUE OF JULY 8TH, 1908 (DR. HERRINGHAM'S CASE).

September 30th, 1908.—She weighs 10 st. 3 lb., as against 7 st. 3 lb. Her abdomen is of almost normal size; the omental ridges have disappeared. Temperature has been practically normal for some weeks, and she is getting about. Appetite good.

Treatment has been tuberculin (T.R.) by the mouth twice a week and mercurial inunctions on the abdomen.

January 14th, 1909.—She weighs nearly 12 st., and has been working as a nurse at a private asylum during the last two months. I have not had an opportunity to examine her abdomen lately. Appetite good. Bowels act normally.

We have been giving tuberculin here by the mouth in a number of cases during the last year. Roughly speaking, those seem to be most benefited where the lesion was peritoneal or intestinal.

Hirschsprung's Disease.—Hellwig reports a case in a young woman who had suffered from birth from obstinate constipation; sometimes intervals of two or four weeks elapsed between defæcation. Serious ileus did not develop until the age of twenty-one, when the anomaly in the intestine was remedied by an anastomosis between the transverse colon and the descending loop of the sigmoid flexure.—*Journ. A. M. A.*, January 2nd.

RECURRENCE AFTER THE OPERATION FOR RADICAL CURE OF INGUINAL HERNIA.*

By W. H. BATTLE, F.R.C.S.

I PROPOSE to limit my remarks entirely to the question of recurrence following operation for the radical cure of *inguinal* hernia in order to elicit the opinion of the Fellows on the question of causation of this recurrence. There can be little doubt that the operation of Bassini is now the operation of choice in this class of hernia when performed in the adult, and although some surgeons have advocated and practise slight changes in the details, I take it that all aim at a restoration of the parts to that state which is normal in the healthy human subject. This implies, if done thoroughly, incision of the aponeurotic wall for the whole length of the inguinal canal, reduction of its contents after opening the sac, and the removal of this from the upper part of the inguinal canal so as to shut off the peritoneal cavity at the internal ring. Sutures of differing material are then inserted behind the cord to approximate the conjoined tendon and internal oblique to Poupart's ligament, and others to close the wall of the canal over the cord and diminish if necessary the external abdominal ring. In the placing of sutures behind the cord that structure is carried outwards, so that it again is placed as obliquely as possible in its course along the canal.

I have purposely said "sutures of differing material," because there is still much variety in the composition of the sutures used; catgut, kangaroo tendon, silk, silkworm-gut, linen thread, etc., all have their advocates. I am ready to admit that each of them possesses some advantage, and there is a great deal to be said in favour of each. Especially is this true of the more quickly absorbed material. If the operation is carried out with such aseptic precautions that primary union is obtained it is a satisfaction to feel that the sutures will not give any trouble in the future, and will quickly disappear, but it is also possible that they may go too quickly, or yield too readily under strain. It is affirmed on good evidence "that the tissues within the grip of permanent sutures of silk, wire and salmon-gut become gradually absorbed even without suppuration, and this clearly leaves the sutured area much weaker than after the use of an absorbable suture like catgut, which does not induce nearly so much of this quick and aseptic necrosis of the tissues, if care be taken not to tie it too tightly." During the time this takes, however, there is no doubt that a considerable amount of fibrous tissue is developed around, and finally in the site of the suture, so that the structures approximated by the suturing are more firmly attached to each other and better able to withstand any unexpected strain. Personally I always use silk. With the general average of recurrence of inguinal hernia after the operations practised I do not wish to deal. It is one of those things that are most difficult to estimate truly, especially in London, where the patients are constantly changing their place of abode, and appear desirous of hiding themselves. Frequently patients cannot be traced after they have left the hospital, and it is useless to try to follow them. A personal inquiry is, indeed, resented, and letters are unanswered. An approximate estimate can be given, but it is subject to correction from time to time, and if we accept the statement of the best results obtained by the operation in the hands of a particular surgeon, we are justified in hoping for a similar success provided we are equally careful in the performance of the operation and in our wound treatment. It is true that our patients in London are not always of the most healthy type, and therefore we may not get such a good all-round record as that obtained where the patients are of a more robust nature, yet suppuration has diminished greatly of recent years, for wound treatment has undergone important changes, and continues to improve.

In his communication to this Society on the subject of radical cure of inguinal hernia in 1906, Mr. Hutchinson, jun., quoted some statistics as to the percentage of cures obtained by himself and other surgeons (vol. xxix, p. 327). Since the date of that paper various statistics have been published by others, but no one has yet equalled the record of Vincenzo ('K. f. Med.,' January, 1908, 'Brit. Med. Journ.,' "Epitome of Current Medical Literature," p. 61), who gives a record of 1500 operations for radical cure by Bassini's method, with one death from ether pneumonia (on the fifth day). He was only able to trace 470 (=505 operations) of his

* Delivered before the Medical Society of London.

cases, and amongst these there were only ten relapses two years after the performance of the operation. It is stated that healing took place by first intention in 1433 out of the 1500 cases.

I have thought it better, therefore, to take a series of cases of operation for recurrence, and consider if any lesson can be learned from them, rather than put before you a percentage of recurrences, which, however correct it may be for the time, is subject to variation according to the success or failure attending a search for the patients, and gives no information about the cause of relapse in particular cases.

Altogether there are 35 operations recorded in my case-books which have been performed for recurrent inguinal hernia,* and of these 34 were required on male subjects, and only one on a female patient. These were almost equally divided between the right and left sides. Eighteen of these had been originally operated on by himself. Of the various operations which had been done it was not possible to find out the exact kind of operation in eleven, the evidence being quite insufficient; but amongst those about which there was no doubt, there had been one MacEwen, eight Banks, fifteen Bassini, but these do not in any way represent the relative numbers operated on by these methods, the number of Bassini operations being far in excess of any others.

It must not be forgotten, however, that the appearance of a hernia in the inguinal region after the operation for radical cure is not necessarily a recurrence of the original hernia. It may be another hernia making its way through quite a different part, and in a series of 107 "recurrent" herniæ (published in the 'St. Thomas's Hospital Reports' for 1898–1907 inclusive) there are two in which it is clearly stated that the new hernia came through to the inner side of the deep epigastric artery, the former hernia having been an indirect one. In each the original operation had been by Banks' method.

The general impression is that suppuration is the most frequent cause for the return of a hernia after attempted radical cure. In only four of these thirty-five operations was there clear evidence that suppuration had followed the original operation; in nine it was not possible to come to a conclusion on this point, whilst in twenty-two there was no doubt that primary union had been obtained. This is all the more noteworthy because all cases presenting the slightest trace of pus are put down under this heading, no matter how slight the amount, whether merely a stitch abscess or a more general suppuration. In the series of 107 from St. Thomas's Hospital, the cases in which it is recorded that there had been suppuration during the treatment of the original hernia only numbered twenty-two. This hardly bears out the statement of Jacobson: "In nearly all the relapsed cases some suppuration had occurred, and this is no doubt the main cause of the relapse."*

There is no doubt that suppuration sometimes acts disastrously by loosening or destroying perishable sutures, and rendering others a source of continued irritation. Still, most of us have probably seen instances in which a satisfactory result has been obtained in spite of our fears—the formation of a large amount of scar tissue binding the parts firmly together.

We all aim at primary union, and are accustomed to get it so much as a matter of routine that we are discouraged if accidental infection takes place, and a more dismal prognosis is apt to be given than circumstances warrant. In Hospital practice we cannot always escape the duty of operating on septic cases, nor avoid having them in the wards afterwards during their treatment; in this way the best record may be spoilt, and the percentage of suppuration unfortunately increased. Again, the organisms that are found in the skin of the patient, especially about the hair-follicles of the pubes, are difficult to expel, and I make it a rule to postpone operation if the marks made by the razor used in the preliminary shaving look red and angry. Of course the razor may have been the means of conveying infection to the skin, but we far more frequently see this condition over the pubes when it has been shaved than in the axilla when that has been prepared in a similar way for operation in breast cases. The hospital barber is also alive to the necessity of thorough cleanliness in his work.

It has been stated that if a hernia is going to relapse, the return will take place within a few months of the date of the first operation. In the

* Of the following age: under 10 . 20 . 30 . 40 . 50
Number of cases . 1 . 10 . 13 . 4 . 7

* Jacobson, vol. ii, p. 64, 5th edition.

series of 35, the period of time varied considerably; in one it happened almost immediately, whilst it was found within twelve months in sixteen patients, between one and two years in eleven, and more than two years in the remainder—eight in number. In other words it returned within twelve months in nearly half the cases.

The actual figures are: Immediate return, 1; 1 week, 1; 2 weeks, 2; 4 weeks, 1; 2 months, 1; 4½ months, 1; 6, 1; 9, 1; 10, 2; 11, 4; 12, 1; 13, 1; 18, 3; 20, 2; 21, 3; 23, 2; years: 2, 3; 2½, 1; 4, 1; 7½, 1; many years, 2.

Inadequate operation may leave the patient exposed to the formation of another kind of hernia on the same side, and until a second operation is performed the nature of this hernia will not be known. The first operation in two instances within my knowledge was, as already said, that of Banks. There is another way in which the operation may be inadequate, and of this there are two examples, one in a female patient, æt. 19 years, and the other in a male adult. We were told that in neither of these could the sac be found, and the operation was completed without its removal, sutures being inserted in a way which would have resulted in success in the case of the female, as return in females after clearing the canal is hardly known, and probably would have brought about a cure in the case of the man.

Obliteration of the sac is undoubtedly an important part of the process of radical cure, and the peritoneum must be shut off smoothly at the internal ring, so that there is no pouching left into which omentum can force its way. I am of opinion that in some of the instances in which recurrence takes place at an early date, there has been a slipping of the ligature after the sac has been cut off, and that the omentum has found the weak spot and passed along the cord. The importance of the presence of the cord is fully recognised by operators, for all will admit that recurrence is hardly known in the female. In addition to the mere presence of the cord, which has a tendency to drag on the inner margin of the internal ring and act as a guide to a descent of peritoneal pouch, it is possible that during the application of the ligature, high in the neck of the sac, some of the cord may be included in the ligature, and that in replacement of parts before the suturing of the wall of the canal this attachment may be pulled upon, and a small pouch formed from the very first. Or again, the dragging of the testis in normal movement of the body may bring this point down at a later date. It may also be that the method of displacement of sac, rather than ligature of sac (before its removal), has the effect of eliminating this cause. The additional sutures placed by some surgeons to close the space above the cord may also effect the same object.

The effect of undue strain is undoubtedly considerable. How much trouble may arise from the vomiting which follows the use of anæsthetics it is not possible to estimate. But the patients not infrequently ascribe their recurrence to the fact that on a certain day they went on horseback, or lifted some heavy weight.

At the end of a fortnight the patient can safely go about again, and walk from place to place in moderation, but the muscular exertion which he undergoes should be gradually increased, and at no time violent. It should be remembered that although the condition of the parts may apparently be as nearly normal as possible, yet the true result of the operation may not be so satisfactory as the operator would fain believe; but given time it will prove satisfactory.

Vincenzo (*op. sup. cit.*) says "that the most usual cause of a relapse, apart from some violent force, is a general laxity and weakness of the abdominal supports. Excess of fat in the abdominal walls is not *per se* a cause of relapse, but as it is generally associated with weak and feeble muscular development, may for the latter effect be followed by prolapse."

In most of the cases which had been operated on by me, as well as in those which had been subjected to operation by other surgeons, there was an excess of scar tissue in the inguinal canal which in some instances matted the parts together in a manner which made it difficult to differentiate the structures. In three instances the posterior wall was so firmly fixed to the cord that Foster's operation was done—that is, the cord was not disturbed, but the internal oblique was sutured to Poupart's ligament over it after removal of the sac, and the canal closed in the usual manner. In eight of the earlier cases the operation was by Banks' method, which was given up by the author as a method of attempted cure eleven years ago.

As regards the condition of the sac, it was fre-

quently difficult to separate, being thickened and adherent to the cord, but in every instance save one it was ultimately separated and ligatured before removal. The exception was made in a case of direct hernia, where the sac presented no definite neck, and was invaginated without the removal of any part of it. In three the hernia had become irreducible, omentum being adherent to the inside of the sac. In two of these omentum required to be excised.

It is interesting to note that tubercle of the sac was present in two patients (æt. 8 and 13 years) associated with tubercle of the peritoneum. At the second operation there was no sign of that disease, but in one there was an unusual amount of fluid present at the time of operation, but not sufficient to give evidence on percussion of the abdomen.

The man who requires an operation after forty years of age, and has been leading a sedentary life with excess of alcohol daily, is not a good subject, and should, I think, be supplied afterwards with a light truss for his constant wear if there is any sign of commencing relapse. As a rule a truss is not required by anyone after the operation by Bassini's method.

To the question as to whether I consider Bassini's operation one which is universally applicable I reply "No," and strongly advocate in herniæ of unusual size the removal of the testis, so that complete obliteration of the canal may be secured—that is, in the adult, where the cord is often much thickened and occupies considerable space.

In cases of congenital hernia in children it may be advisable to take away an atrophied testis which cannot be brought down, but if it can be managed it should be placed in the scrotum and retained there during the process of healing by some effective means. The importance of this may be considerable in later life if the patient wishes to enter the services.

Mr. J. Hutchinson, jun., in his paper advocated removal of the testis in recurrent cases, and although I am not in favour of that addition to the operation in the majority, two cases which have been under my treatment have made me regret that their consent to this had not been asked. They are cases in which none of the usual causes of recurrence could be found; they were strong healthy men, and on each occasion operation was followed by primary union. I hope that they will not require any further operation; if they do they will be advised to that effect. Perhaps in despair of help from me they have already been put right by one of the Fellows of the Society.

I think that they are worthy of special mention, for they showed a disappointing tendency to recur.

The first is that of a sailor, æt. 35, a strong, muscular man.

The primary hernia was a reducible left inguinal hernia of three months' duration, for which Bassini's operation was performed by me in October, 1895.

Recurrence took place fourteen months later, and a second operation by Bassini's method was done in January, 1897.

Recurrence followed thirteen months later, and a third operation was performed in June, 1900.

The hernia reappeared after three months, and a fourth operation was performed in July, 1902. At this operation the sac was noticed to be of unusual thickness.

Primary union followed each operation.

The second is that of a soldier, æt. 25 years, who had a left inguinal hernia, the result of a fall. For this two operations were performed in India in 1902. Suppuration appears to have followed on each occasion, and almost immediate return of the hernia was reported.

Bassini's operation was done on July 12th, 1904, and a month later the hernia reappeared.

A similar operation was performed October 7th, 1904. On both occasions primary union resulted.

I do not wish the Society to imagine that I am putting forward these eighteen cases of recurrence from my cases as a complete list of the whole of my recurrences. Some have refused operation and are using trusses. These are all that have applied for a second operation out of about a thousand cases of inguinal hernia that I have had to treat.

Some of the Fellows of the Society may remember a statement made by Mr. John Langton during the course of a discussion on the subject of hernia some years ago at this Society, to the effect that he had a book in which he had entered the cases of recurrent hernia that had come before him at the Truss Society. He implied that many of us would be surprised to find our names in the book, or words to that effect. I hope that some day he will give us the results of his notes on this subject; they would be very instructive. And I may add that as we want to find out the truth about the

subject the profession would be indebted to him. They must, however, include many cases of recurrence of the operation by methods which were in use during the earlier days of the operation for the radical cure and have now been abandoned. If we may draw conclusions favourable to the operation from the numbers who apply at the hospital for it, then it confers the benefits which some of us hope it does confer, for the numbers are large and increase yearly. Quite a tax is made on the beds at our disposal and on our time. Only recently I had to perform ten of these operations on the same afternoon.

There is still an impression amongst some members of the profession that the operation is a serious one, and one still comes across cases in advanced life to whom a hernia is a constant annoyance, pain and source of danger. This ought not to be, and need not be. We are not endeavouring to hide our results, but to put them forward fully in hopes that we may obtain better.

Mr. TURNER said he had himself performed the operation over 500 times, and in 1904 he wrote to all the hospital patients upon whom he had at that date performed the operation, namely 250. Just about half that number replied, and out of those 125 he saw 122 at the hospital. There were recurrences in three of them. One of them was a man who came to him after having been operated upon for hernia first, so that his own operation was the second. Another was a man who was sent to a convalescent home, and who, against instructions, got up and walked about the grounds, as his wound was healed. Although he had those three recurrences in his own cases he thought it was not right to argue that that was the right percentage, because, as Mr. Battle had already mentioned, the recurrences often went elsewhere for advice the second time. It was not unusual to find hernia develop on the side opposite to that operated upon; he had seen at least a dozen such cases. The operation he did was a modified Bassini. It was impossible to say what was the main cause of recurrence. Even the coming away of stitches did not always cause it. A short time ago he saw an elderly gentleman who had been operated upon for hernia one and a half years ago, in whom a modified Bassini was done, and, though he had lost seven out of his eight stitches, the hernia appeared to be cured. Even violent coughing did not cause bulging of the abdomen. It was only fair to remember that the surgeon who operated sometimes did not expect a cure. He had himself operated upon several cases in regard to which he could not say he expected a cure in the sense that it would not be necessary for the patient any longer to wear a truss, and if such a case as that came back to the hospital it was scarcely fair to regard it as an actual recurrence. He did not think it was very important what material was used for sutures. He himself never used silk. In the old days, when it was not so well sterilised as now, one used constantly to see sinuses leading down to buried silk stitches, and in a convalescent home, where he was surgeon, he had seen several such cases. Mr. Battle appeared to be rather diffident in operating upon people over forty years of age; personally, he (Mr. Turner) never let the age of the patient stand in the way, unless it was such as to make it undesirable for him to have a general anæsthetic. If the patient were an old man with bronchitis and emphysema and various lung complications which made it inadvisable to give him chloroform or ether, it was inadvisable to operate upon him. Not long ago he operated upon a man who was officer in the Indian Mutiny, so that he must be well over seventy years of age. He had good lungs and made an excellent recovery. He did not think removal of the testes was ever necessary in the operation unless the patient were an adult who had a rudimentary testis which was never likely to be of any use to him. He did not practise removal of testes. He did not think it was necessary to be careful that the sac of the hernia should be removed flush with the peritoneum, so that there should be no pouch; if the cord was well pulled upon when it was cut off it mattered very little whether there was a depression on the peritoneal surface or not. He did not think it could have anything to do with the recurrence of a hernia. He agreed that if there was a septic inflammation of the parts one would naturally put off the operation, yet it was curious, even when there was an incision made through a patch of chronic eczema, the wound would do very well.

Mr. ARTHUR BARKER said all had doubtless had their troubles more or less in connection with radical cure of hernia, and he did not think it was

possible to fix any percentage of recurrences with perfect accuracy. In over two hundred cases he tried to keep a record and follow out the subsequent course, by having a little book for each, in which was entered the particulars of the case. But there were always some who escaped being traced for long periods, and as they might have been just the ones in which there was recurrence, that vitiated any conclusions which might be sought to be drawn from them. He gathered that Mr. Battle's taste in regard to sutures was fairly catholic, but that he relied chiefly on silk, which had been his own practice for many years. But every kind of suture would come out sometimes, not necessarily because of sepsis, but sometimes because the sutures were tied too tightly. He believed that one of the reasons fewer recurrences were seen nowadays was that the practice of tying tissues very tightly had been largely given up. It was now recognised that it resulted in strangling and necrosis of those tissues, and this brought the stitches to the surface if it did not actually determine the recurrence. The deep follicles of the pubes would sometimes prevent that blameless aseptic healing which all so much desired. An important point, which was not touched on, was that of recurrences where the tissues dealt with were obviously below par as to development; in such cases he thought it was often advisable to use a wire network in order to support the parts. It certainly made it unnecessary to draw the fascial structures together so tightly. He had had encouraging results from the insertion of a silver network, which could be put in under the external oblique and the cord. This would also obviate the removal of the testes in some cases. He would like to hear from Mr. Battle whether he had used such a device, and if so with what result. His own view was that there was an opening for such supports.

The PRESIDENT expressed his appreciation of the manner in which Mr. Battle had brought forward the subject, and of the remarks which had been made by Mr. Turner and Mr. Barker. The paper and subsequent discussion showed very clearly the astonishing safety of the operation. He understood Mr. Battle to say that 1500 cases had been recorded, with only one death, and even that might have been due to the anæsthetic. The frequency of recurrence had also been shown to be enormously diminished of late. He had always thought that the statements made by surgeons connected with the Truss Society were somewhat biassed, because they obviously saw the cases which recurred, not those which did not recur, and that led to their having an exaggerated idea of the frequency of recurrence. The discussion also appeared to show that much of the diminution of recurrence was due to the careful selection of the cases for the operation, and to the greater skill and efficiency with which the operation was performed. There was also a greater general skill in the organisation of such operations. His own observation led him to think that if recurrence was going to occur it would do so within the first year from the primary operation.

Mr. BATTLE, in reply, said he was fortunate in having to bring the matter before the Society when Mr. Lockwood, who had such a large experience in such matters, was its President. He asked Mr. Turner after what period from the operation he saw the 122 cases which came up to the hospital to show themselves, and whether he formed an average period in arriving at his conclusions?

Mr. TURNER replied that the period varied from eighteen years downwards, but none of them were recent cases.

Mr. BATTLE, continuing, said Mr. Turner appeared to have misunderstood him with regard to age, as he spoke of forty years of age in connection with recurrences, not the original duration. He had himself operated on patients up to the age of seventy-two. With regard to silver wire net-work as a means of support, he was very averse to using any such apparatus, especially in a region where there was so much movement. He had known cases where such apparatus had done much damage and caused troublesome suppuration, and in one case it apparently produced ulceration into the underlying bowel, and the patient reappeared at the hospital with a fæcal fistula. With regard to removal of the testis, it was only in cases where the radical cure was exceptionally difficult, where the hernia was large and the patient was getting on in life, that he had practised removal of the testis. Mr. Hutchinson, jun., recommended its removal in most cases of recurrence.

February 1st, 1909.

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A CLINICAL LECTURE

ON

APHASIA.*

By GUTHRIE RANKIN, M.D., F.R.C.P.,

Physician to the Dreadnought and Royal Waterloo Hospitals.

IN one of the wards on upper east floor there is a patient whom many of you have seen from time to time; J. W—, æt. 45 years. By occupation he was a sailor, and he was admitted to this hospital as long ago as the year 1898. He is one of our naval pensioners, and is entirely confined to bed on account of a right hemiplegia with spastic contraction of both upper and lower limbs on the right side. His case is one of ordinary hemiplegia, probably from a specific cause, and is somewhat out of the ordinary run of such cases from the fact that the patient suffers periodically from typical attacks of Jacksonian epilepsy. The main interest in his case, however, so far as we are interested in him to-day, is that he is speechless. This loss of the power of speech came upon him after his first attack of apoplexy as long ago as 1890, and, though he understands perfectly everything that is said to him, and can also read intelligently both printed and written language, he is quite unable to utter more than a few stray words intelligibly; not because he has any impairment of power in his articulatory muscles, but because his original cerebral lesion has involved either his motor speech centre in Broca's lobe or his pyramidal tract lower down, in the internal capsule or elsewhere, which contains, as you will remember, the fibres connecting Broca's lobe with the lowest centres in the medulla. The patient is, in fact, a victim of the motor variety of aphasia.

By the courtesy of Sir Dyce Duckworth I am also able to bring before you a patient from one of

* Delivered at the London School of Clinical Medicine.

his wards, in whom the symptoms of one of the sensory varieties of aphasia are graphically portrayed. This man is 47 years of age, and some months ago he was attacked, like the other, with right hemiplegia. The muscular palsy has been fairly well recovered from, though evidence of it is still obviously enough present, but the disability which remains from his cerebral lesion, and which so seriously troubles him now, is his inability to express himself in intelligible language. The fault does not consist, as in Walker's case, in a powerlessness to use words. He can speak volubly, but his vocabulary is limited, and his appreciation of the meaning of the words he uses is defective. Hall understands all that is said to him, but, though fairly educated, he cannot read either print or writing. He is able to write his own name, but nothing else, as frequently happens in such cases, but he is quite incapable of reading the name after it is written. He has also lost his memory for words, so much so that, though he can write it, he cannot remember his own name so as to speak it. His original lesion has involved his visual word centre together with some of the commissural fibres by which it is connected with other centres concerned in the production of coherent speech, and he is suffering from that form of sensory aphasia which primarily depends upon the occurrence of word-blindness.

Speech is a function which is gradually acquired by imitation. The centres upon which its mechanism depends are situated both in the cortex and in the medulla. Disturbance of the cortical centres causes aphasia in the widest acceptation of the term; a lesion of the medullary centres produces anarthria. In motor aphasia, such as is manifested in Walker's case, the intelligent manner and gestures of the patient show that, though unable to give verbal expression to his thoughts, he is capable of thinking, and the occasional distinct pronunciation of one or more words proves that the speechless condition is not caused by a mere mechanical difficulty in the power of articulation. The few words which the aphasic patient can utter are always clear and distinct, in this respect differing markedly from the blurred thickness of speech which characterises bulbar paralysis or alcoholism.

Loss of power of speech may be associated with more or less loss of power of writing—agraphia. In the presence of a right hemiplegia, the patient may be unable to write because of the paralysis of his hand muscles, but agraphia is also frequently present as an element in aphasia and as a direct result of the central lesion, as in the second case.

The power of speech depends upon two sets of processes—sensory, or in-going, and motor, or out-going. The senses of hearing and sight, and, in the blind of touch, are the pathways by which the materials for speech reach the sensory centres; from these the impressions are reflected to emissive centres which govern spoken and written language, and these again in their turn regulate lower centres in the pons and medulla which control the movements of the tongue, lips, and larynx in speaking, and of the hand muscles in writing.

In addition to these two receptive and two emissive centres, the existence of a higher or psychical centre capable of generating independent impulses, which find expression in spoken or written words, has been promulgated by Broadbent, Lichtheim, Wernicke and others, but most observers now agree that the supposition of such an ideational centre is unnecessary. Bastian contests the theory of true motor centres in the cortex. The two centres which we are accustomed to regard and speak of as motor are, according to him, though motor in effect, really sensory or kinæsthetic in their true character. It is important to remember that all motor processes are reflex acts in that they are originated from the sensory side of the nervous system, and that our only consciousness of them is the appreciation of the sensations which accompany their performance. When, for instance, words are learned, the sensations accompanying their articulation and phonation are located in a centre which, though for convenience called motor, is more accurately designated, in accordance with Bastian's nomenclature, the "glosso-kinæsthetic centre." Similarly, when the power of writing is acquired, the sensations accompanying the movements of the hand are registered in the other so-called motor centre, to which the more accurate name has been given by him of "cheiro-kinæsthetic centre." Both these kinæsthetic centres are subservient to their corresponding sensory or receptive centres, and never act alone; the glosso-kinæsthetic centre depends upon the auditory word centre for its education and incitation to activity, and the cheiro-kinæsthetic centre is simi-

larly dependent upon the visual word centre for its stimulus to action.

In right-handed persons these four speech centres are situated in the cortical grey matter of the left cerebral hemisphere.

Of the sensory or perceptive centres, that in relation to the sense of hearing is the most important. The auditory word centre is the first pathway by which the child receives impressions in the education of his speech processes, and in the majority of persons it remains the dominant speech centre throughout life. It is situated in the posterior two thirds of the first temporal convolution, and if this region becomes the seat of disease there ensues inability to understand the meaning of spoken words, although they may still be heard as meaningless sounds. To this defect in the mechanism of speech the term word-deafness is applied.

The perceptive centre which is related to the sense of sight is situated in the angular and supramarginal gyri, and when it is impaired by disease the patient is unable to read or recognise printed and written words. This condition is known as word-blindness.

The motor, or kinæsthetic, centres are situated: That for speech—the glosso-kinæsthetic centre—in the posterior part of the third frontal convolution, Broca's lobe; and that for writing—the cheiro-kinæsthetic centre—query, in the corresponding situation of the second frontal convolution. It is still doubtful whether the speech and writing centres are separate, and though there is much evidence in favour of their independence of one another, Déjerine and others still believe that both are represented in the one centre in Broca's lobe. The speech centres are freely connected with one another by commissural fibres, and these serve to connect the various memory pictures which combine to form the word-image. It is only by such an association that a true concept can be formed and expressed in intelligible written or spoken symbols: so that a lesion in the association fibres by which the perceptive and emissive centres intercommunicate, will result in defects of language characterised by an unintelligible use of words or by an inability to recall the names of things seen or heard, or by other speech defects of various types.

Each cortical centre is responsible for a special kind of word-memory, and it is important to recognise that the four principal varieties of word-memory are mainly associated in couples. The auditory and glosso-kinæsthetic memories are brought into play in ordinary speech, and the visual and cheiro-kinæsthetic memories in ordinary writing. "In expressing oneself in spoken words," says Bastian, "the memories of such words are first principally revived in the auditory centre, and then the nerve-units thus called into activity rouse in immediate succession the corresponding glosso-kinæsthetic elements before the pronunciation of the word can be effected through the aid of the motor centres in the medulla. Similarly, when expressing our thought by writing, though the memories of words are probably first revived in the auditory word centre, like memories are almost simultaneously revived, through the intervention of the audito-visual commissure, in related parts of the visual word centre; and from this region stimuli must pass through corresponding cheiro-kinæsthetic elements before the actual writing of the word can be effected through the instrumentality of motor centres in the cervical region of the cord."

The functioning connection between the different centres is so intimate that an extensive lesion of one always causes more or less disturbance of the others. It is seldom that the lesion causing aphasia is limited to one area; it generally overflows so as to involve commissural fibres as well as speech centres.

The executive centres for articulate speech are situated in the floor of the fourth ventricle, while those for writing are situated in the anterior cornua of the cervical enlargement of the cord. Both are connected with their respective kinæsthetic centres by a band of fibres in the pyramidal system.

It is well to remember that asphasia may be produced by functional as well as by organic disease of the brain. A right-sided convulsion is often followed by transient inability to speak, and not infrequently the congestive attacks to which general paralytics are liable produce temporary aphasia. Hysteria, or intense emotion, may also give rise to such an inhibition of function as to render the speech-centres temporarily unable to transmit sufficient energy to excite activity in the nuclei of the executive nerves.

From what has been said it will be apparent that,

according to the situation of the causative lesion, aphasia may be either sensory or motor in type. The damage done to the brain may implicate one or more of the cortical centres, or the commissural fibres between them rather than the centres themselves may be principally involved. There is thus room for a wide variety of aphasic manifestations, but time will only permit of a short reference to the more important of them.

(1) *Sensory aphasia.*—When this form of aphasia is complete the patient's consciousness fails to respond to the evidence of his senses. The mind is blind, or deaf, or otherwise insensible to objects which were formerly familiar. The two most common and important defects are word-deafness and word-blindness.

Word-deafness is always associated with a lesion of the first left temporal convolution. A patient suffering from this variety of sensory aphasia can hear ordinary sounds but is unable to recognise spoken words. He is able to write, but not, on account of his word-deafness, to dictation. Like a parrot, he may repeat words or sentences, but they awaken in his intelligence no auditory memories, and so are incomprehensible to him. He is in the condition of a man who listens to a foreign language; he hears the words but is unable to attach any meaning to them. If there is no accompanying word-blindness, he may be able to read both aloud and to himself, but the visual word centre is in such close association with the auditory word centre and is so much under its guidance that very frequently the perception and expression of written speech becomes impaired from secondary depression of the visual centre's functional activity. Similarly, the glosso-kinæsthetic centre, when it loses the controlling influence of the auditory centre, is apt soon to become hopelessly unable of itself to supply rational speech. The exception to this general rule is found in those comparatively rare persons whose memorial processes take place mostly through visual centre impressions and not through auditory centre impressions, as is the general rule. When the lesion of the auditory word centre is incomplete, the word deafness may be partial only. Under such circumstances the patient may recognise the sound of his own name, though deaf to all other words. It would seem as though its familiarity enabled it to awaken recognition in cells wholly irresponsive to the excitation of any other spoken language. He may be capable of giving automatic expression to a considerable vocabulary, especially of swear words, but quite unable to recall the proper words for the expression of thought. He substitutes wrong words for those he wishes to employ, but he is unaware of the errors he commits, and he often has recourse to a roundabout phrase to express the particular word he is unable to remember. This form of speech defect, which is known as amnesia, is familiar to us all as occurring in a mild degree in old age, or during convalescence from a long and serious illness. Under such circumstances a person is often observed to have become strangely forgetful of certain words, and it is remarkable that proper names and nouns are more readily forgotten than verbs and adjectives.

Word-blindness may occur either alone or as an accompaniment of motor aphasia. The patient can neither see nor understand printed or written words; they appear like hieroglyphics, and fail entirely to recall to his memory the meanings which they represent. If the lesion is confined to the visual word centre speech is not seriously interfered with, because both the receptive pathway through the auditory centre and the expressive pathway through the glosso-kinæsthetic centre remain unimpaired, but it necessarily entails inability to read aloud or to copy print into writing. The visual centre dominates, and to a great extent controls, as you will remember, the cheiro-kinæsthetic centre, so it is not surprising to find that in cases of word-blindness there is generally an accompaniment of agraphia. If the lesion is not extensive it may cause partial word-blindness only. Under such circumstances the patient may retain the power of recognising his own name in print or writing, though unable to read any other word; or he may be able to recognise numerals, and to add or multiply simple numbers correctly though he fails to identify letters or words. Patients who are word-blind can sometimes write to dictation or from copy, but they are unable to understand or to read what they have written. The proximity of the auditory and visual word centres renders them liable to simultaneous damage in the neighbourhood of the posterior end of the Sylvian fissure. Should the lesion be thus extensive, word-deafness and aphasia become associated with word-blindness and agraphia, and the only power of

understanding left is through the medium of gestures.

(2) *Motor aphasia.*—This variety of aphasia was first recognised by Broca, and the lesion upon which it depends is situated in the lower end of the third left frontal convolution. The patient with motor aphasia has lost his memory for the efforts which are necessary to produce articulate speech. In simple cases he can read to himself, though not aloud, and he understands all that is said to him. He may be unable to utter a single syllable, but more frequently he has command of a few simple words such as "Yes" or "No," or of a short expression or phrase such as "Oh my" or "Dear me," which he is apt to repeat whenever he tries to speak. Under the influence of emotion, he may suddenly ejaculate one or more words which are impossible for him when quiet. He understands all that is said to him, though unable to repeat it, and by gesture he can demonstrate the uses of various articles, though he is unable to name them. If, for example, he is shown a pencil, and he be asked successively whether it is a knife, a coin, a corkscrew, or what not, he will instantly shake his head in disapproval, but when the question is finally put, "Is it a pencil?" an intelligent smile and appreciative nod will at once prove that he both knows what the article is and appreciates the meaning of the word which describes it. If it is within his power to articulate any words at all, he is fully conscious of the mistakes he commits when the words he uses are wrong, and in this respect he differs markedly from the patient suffering from the sensory form of word-deafness, who, as has been already mentioned, is also liable to use wrong words, but without knowledge of his error. Agraphia, though a frequent, is not an invariable concomitant of motor aphasia. The faculty of writing is acquired by the child long after it has learned to read; his graphic centre is trained through all the other centres, and therefore in every form of aphasia there is a liability to agraphia, though it is most complete in cases of word-blindness.

Motor aphasia, when once established, may remain permanently, but sometimes by a slow and tedious process of education of the right hemisphere a considerable power of speech may be regained. In the right hemisphere there exist similar speech centres to those present in the left hemisphere, but they lie fallow in ordinary right-handed persons. In those who are left-handed the right hemisphere of the brain is the originally educated hemisphere, in contra-distinction to the left hemisphere in those who are right-handed. The two hemispheres are intimately connected with one another by commissural fibres in the corpus callosum, through which the dormant centres on either side may be stimulated and made active.

In addition to the defects in speech, which result from a lesion situated in one or other of the speech centres, other difficulties may arise from interruption of the association fibres, by which these centres are connected with one another, and with the executive centres in the medulla. When the tracts between the receptive and emissive centres are severed a condition of aphasia ensues, which is characterised by inability to recall the name of a thing seen or the appearance of a thing named, though the thing itself is quite clearly appreciated mentally when named or exposed to view. The memory picture is not lost, written and spoken words being both understood perfectly, but the impulse started in either the auditory or visual centre is interrupted somewhere in its progress from the cortex towards the medulla, and the speech mechanism is so dislocated that the correct spoken or written expression of the concept is impossible. The association fibres joining the various centres are as necessary to the proper use of even a single word as the centres themselves, and the result of their disturbance is either a misuse or a total inability to use words similar to that produced by a lesion in the glosso-kinæsthetic centre itself.

The speech defects which ensue upon structural or functional disorder of the communicating fibres passing in the pyramidal tracts from the kinæsthetic to the executive centres differ from those which arise in the bulbar centres themselves in that they are unassociated with difficulty in deglutition and other symptoms characteristic of true bulbar paralysis. The speech imperfection is obviously not a sensory defect; the subjects of it can revive the memory of words, and are able both to think and to express their thoughts in writing. Every simple word spoken is understood, and attempts are made to repeat it; whereas the patient whose auditory word centre is inoperative is unable to understand any spoken word or sentence, and therefore makes

no attempt at repetition. The cortical centres in which the memory of words is stored are intact, so that there is nothing to interfere with the flow of thought; the damage is subcortical, and involves only the functional integrity of the lowest or executive centres.

It would conduce to much greater clearness if Bastian's suggestions as to nomenclature were generally adopted. He would restrict the term "*aphasia*" to the defects of speech produced by lesions in Broca's lobe; the term "*aphemia*" to those produced by subcortical lesions in the pyramidal fibres; and the term "*amnesia*" to those produced in the auditory and visual word centres, and in the commissures by which these are connected with one another and with corresponding centres in the opposite hemisphere.

Asphasia presents many complex phenomena, the sensory and motor types of the condition being capable of intermixture in innumerable varieties of combination. A little patience will serve to unravel the difficulties, and enable the situation of the lesion to be diagnosed accurately in the majority of cases, if we remember that complete mind-blindness and mind-deafness result from a lesion involving both auditory and visual centres; that word-deafness results from a lesion in the first left temporal gyrus; that word-blindness results from a lesion in the left angular and supra-marginal gyri; that motor asphasia results from a lesion in the lower end of the third left frontal convolution; and that paraphasia, in all its many varieties, results from a lesion in the association tracts which unite the sensory with the motor and executive centres.

February 8th, 1909.

Cancer.—Park insists that cancer is a specific disease, intensely infectious and transmissible, being caused by an extrinsic agent. He therefore urges that cancer must be treated much as we would treat a filth disease, especially when it has advanced to the ulcerative stage—namely, by the most efficient cleanliness. It would be well, also, to adopt Brand's suggestion that no food should be prepared or consumed with unwashed hands. In addition to this, it would appear that in some respects, at least, we ought to be at an advantage in treating this disease, since its earliest manifestations are local.—*Journ. A. M. A.*, vol. lii, No. 3.

SOME CONSIDERATIONS OF THE TREATMENT OF DISPLACEMENT OF THE ABDOMINAL ORGANS.*

By WILLIAM SHEEN, M.S., F.R.C.S.,

Surgeon to the Cardiff Infirmary; Consulting Surgeon to the Seamen's Hospital, Cardiff.

THE treatment of the displacement of abdominal organs may be divided into two classes, operative and non-operative, and I shall venture, even at the risk of being thought to trespass outside my province, to say a little about not only the operative but also the non-operative treatment. For it is in just this class of case that the surgeon must be fully satisfied that non-operative measures have had a full and proper trial. The surgeon is not merely a carpenter, but should have presented to him all the aspects of the case, and have a first-hand knowledge of the signs and symptoms present before determining upon the advisability of operation, otherwise he can, of course, take no responsibility in regard to beneficial results.

These remarks apply particularly to the class of case under discussion. The patients, the majority of whom are women, are almost always highly neurasthenic. The degree of the abnormal physical signs is no measure of the severity of the symptoms. Symptoms with regard to a particular organ may only arise when its abnormal condition is discovered by a doctor. Relief and often apparent cure may follow an operation which has been purely of an exploratory character. Various diseases are from time to time diagnosed as one or other symptom becomes a prominent factor. So, for example, the same patient may have accumulated the following string of diagnoses: gastric ulcer, gall-stones, appendicitis, colitis, right movable kidney, and displaced uterus. Similarly this patient may have had a choice of all the following operations: gastro-enterostomy, cholecystostomy, ileo-colostomy, appendicectomy, appendicostomy, hysteropexy, and nephropexy. One, two or three of the operations may have been performed. The operation is frequently "successful," but far less often is the patient "cured." The mechanics of the surgeon cannot restore to the normal the perverted nervous mechanism of the patient.

* Read before the Newport, Monmouthshire, Medical Society.

This is a class of case over which physician and surgeon must unite: the knowledge of the patient possessed, and possessed alone by the family doctor, is often of inestimable value. Where, perhaps, half the pharmacopœia has been run through, where "cures" innumerable have been tried, where patent pills and electric belts have each achieved a fleeting success, it would be rash indeed for the surgeon to prophesy a speedy and complete cure by fixing a movable kidney or raising a dropped stomach.

The normal position of organs.—It is proper to consider this just for a moment with regard to abdominal organs before passing on to study of the treatment of their displacements. One wishes not only to know these normal positions, but also what deviations from the normal are permissible comparable with the maintenance of health; what, in fact, are the "normal abnormalities," as Goodhart wisely calls them. Not only abnormalities of structure, but abnormalities of function must be considered. For information about abnormalities of structure I have consulted the anatomy books, but I find them far less precise and detailed when dealing with the viscera than when, for example, with an artery or a muscle. However, one can obtain a certain amount of valuable information as to the variations in position and size of an organ within the limits of health, and for this information I am particularly indebted to the article by the late Professor Birmingham on "Digestive Organs," in Cunningham's 'Text-Book of Anatomy.' These variations I will speak of when dealing with the organs separately.

Abnormalities of function must also be remembered. With one individual it may be "normal" for defæcation to take place once a week, with another to vomit daily, with another to ruminate.

How are the viscera maintained in their position in the abdominal cavity? We read much in the anatomy books of the various ligaments and peritoneal folds which support the different organs, but, in my opinion, these have little to do with maintaining the viscera tightly and firmly in their positions. The ligaments are more like anchor-chains, they limit the movements of organs, but do not fix them rigidly in one place. The main factor in keeping the abdominal organs in position is atmospheric pressure, comparable to the way in which atmospheric pressure keeps the head of the femur in the acetabulum. The abdomen is a closed cavity, and contains no air, and no free space of any kind, and all the organs are tightly packed together within it. What happens, then, when organs, such as the bowel, the bladder, or the stomach empty themselves, and when constant variations in calibre of the alimentary canal take place as contents pass along? This is a question difficult to answer. Partly variations in capacity are due to relaxation or contraction of the abdominal muscles, partly diminution in the calibre of an organ is compensated for by movements of gases in adjacent viscera.

Consider an organ like the liver. The liver is the heaviest organ in the body, and yet it lies at the top of the abdomen, and in spite of the vertical posture of man, rarely gets displaced therefrom. Its ligaments do not hold it there, the falciform ligament is found quite lax on opening the abdomen. It is held in position by atmospheric pressure. Just as a tooth-plate is held in position against the hard palate and jaws, and as on the tooth-plate all the grooves and ridges of the palate are reproduced, so the liver is held in position against the diaphragm, and on its upper surface we find grooves and ridges representing all the structures with which it comes in contact. The liver may be regarded, not as the first structure under the roof of the abdominal cavity, but as part of the roof itself.

Similar considerations enable us largely to understand the constancy of position of other organs, and the method of preparing bodies by formalin hardening has thrown much light upon the natural shape and position of the different viscera, and of the grooves and ridges on them.

That abdominal organs may stand much displacement without disturbance of function is shown daily by the finding of an organ carrying on its functions quite normally under apparently most trying conditions of situation and pressure, due to clothing, occupation, increase or decrease of fat, pregnancy, tumours and so on.

I feel that I have kept you over long with these general considerations, and will now pass on to the treatment of displacements of the different organs considered separately.

Kidney.—This is the most ancient example of a moving abdominal organ. A distinction is sometimes made between a "movable" and a "float-

ing" kidney, the latter being supposed to have a mesonephron. Such a kidney has, as far as I know, never been seen. The mobility is always *behind* the peritoneum. The majority of people who have moving kidneys do not know that they have got them, and are not troubled by them. In the minority symptoms occur, but, because there is movable kidney and because there are symptoms, we must by no means be assured that the symptoms are due to the movable kidney. The movable kidney is often only one of the items in a general visceral proptosis, and so the position of every organ must always as far as possible be ascertained. The best practical test that I know as to whether a movable kidney is really the cause of the symptoms is the test of rest. If the pains, etc., cease or are relieved at night it is likely that the kidney condition is at the root of the trouble. Having got thus far, the necessity for operative treatment can be discussed, and here one finds nowadays that patients and doctors alike are usually too prone to jump at operative treatment. Rest is condemned as tedious, and belts as cumbersome. At the same time I will venture to assert that a properly fitted belt, the application of which is preceded by a couple of months' rest cure, with abdominal massage, will give as much relief to the majority of patients as any operation. The preliminary treatment restores fat and muscular tone. The belt to be useful must be a properly constructed one such as that described by Sir Frederick Treves in the 'Practitioner' for 1905. The oval pad sewn on the inside of a piece of webbing is of absolutely no use. Treves records about 300 cases treated by his belt, in the majority of whom great relief or cure resulted. There will remain, however, a number of cases in which operation is necessary, and here again the question will be asked, Does operation afford a permanent cure? By absorption or loosening of the stitches will not again the kidney become loose, or will it not be necessary to wear a belt after operation to obviate the possibility of such loosening? These are very pertinent questions. I believe that the kidney can be so anchored as to fix it permanently in position, and that no belt or other support will be required after operation. I base this belief on correspondence with a number of patients upon whom I have operated, and also upon an autopsy I obtained some years ago upon a patient who died of phthisis about six months after a nephropexy. In this case the organ was firmly fixed in position, and the whole of the track, where it had previously oscillated up and down to the iliac fossa, could be plainly seen. The method I adopt is to expose the kidney through the usual oblique incision, to pull it entirely from the wound, and examine it and its pelvis for any disease such as tubercle, or any evidence of kinking of the ureter or hydronephrosis, and then to replace it and fix it by silk stitches passed on a curved, blunt-pointed, round-bodied needle going through the fascia and the kidney substance. About six stitches are used, and on tying them the kidney is fixed, and the deeper parts of the wound closed at one and the same time. I have never been able to anchor satisfactorily by means of the capsule, which I find too thin.

The main indications for operation are evidence of a very movable kidney, a painful kidney, so that the belt cannot be worn, the occurrence of Dietl's crises, hydronephrosis, and the suspicion of disease in the kidney.

Anterior nephropexy.—On two occasions I have fixed the kidney through an anterior incision which opened the abdomen. One of these was in a case of hydronephrosis, and the other, to which I more particularly refer, a case of general visceral proptosis. The posterior peritoneum is incised over the kidney, the organ freed, pushed as much as possible into position, and stitches passed through its substance as in ordinary nephropexy and through the lateral parietes as far back as possible. When tied, these stitches fix the kidney and partly close the posterior peritoneal wound.

"*Insanity cured by a new treatment.*"—This must have caught the eye of most of us as it appeared week by week on the front cover of the 'British Medical Journal,' and one was mildly astonished to find that the new treatment was only our old friend nephropexy. The title of this contribution to medical literature is somewhat misleading, but the writer, Dr. Suckling, makes out a fair case in concluding that female patients with melancholic symptoms and movable kidneys lose the former when the latter are fixed. I myself was asked recently by Dr. Cyril Lewis, of Cardiff, to fix a very movable kidney in a lady under his care, a patient definitely melancholic, with delusions of being a lost soul, and forsaken by God. The

immediate result of the operation was the disappearance of these symptoms, but the case is too recent to justify its being called a "cure."

Liver.—The liver may be displaced in man as well as in woman. Its displacement, as might be foreseen, is usually accompanied by displacements of the stomach and of the transverse colon. Where the condition is one of general visceral proptosis, the rest cure, followed by a properly-fitting belt and general measures, usually gives relief, but a movable liver may be found when operating for other more definite conditions, and should then very properly be fixed. I have had several cases of this kind, particularly cases of gall-stones, where the mobility of the liver has been so great that I have been able to lift part of the right lobe of the liver with the gall-bladder right out of the abdomen, having on several occasions performed a cholecystectomy extra-abdominally. I believe that such a movable liver, by reason of its causing kinking of the cystic or bile duct, may be the cause of gall-stones. After the gall-bladder operation, I have fixed the liver by silk stitches passing through the falciform ligament centrally and the liver substance laterally, and going through the peritoneum and fascia of the abdominal wall. In another class of case, where the symptoms have been mainly gastric in type, I have fixed the liver, and by such fixing have raised the stomach; and in other cases I have not only fixed the liver but pleated the small and great omenta so as to raise the stomach and transverse colon respectively. Time prevents me giving details, but I have had these liver cases under my notice for over a year after operation, with entire relief of their symptoms.

Stomach. — Gastroptosis is perhaps the most marked and familiar feature of "Glenard's disease." The stomach, slung up at its cardiac and pyloric ends, sags down between these points so that the aorta, and the pancreas can be felt through the thin and stretched small omentum. We have to consider whether the stomach is displaced or dilated or both, and also whether atony of any degree is present. Usually these three abnormalities are associated, each in a great or less degree. The anatomists tell us that in extreme distension the lowest portion of the stomach does not descend below the sub-costal plane, that its greatest length is 10 to 11 inches and its greatest breadth 4 to 4½ inches, and that it holds about a quart. When empty and retaining its tone it contracts into a cylindrical bag like a piece of thickened small intestine. The transverse colon floats up in front of it so as to lie between it and the parietes. All these facts should be borne in mind in opening the abdomen for possible stomach trouble, and further, no operation should be performed until the proper chemical and other investigations of the stomach contents and stomach digestive powers have been conducted. Non-operative measures of restoring a dropped stomach consist in rest, massage and dieting, and most remarkable results have been obtained by these means, the stomach rising to, and maintaining, a much higher level than, formerly. Subsequently support can be afforded by an abdominal belt. If these measures fail, and more often because the abdomen is quite justifiably opened from suspicion of organic stomach disease and only dropped stomach is found, operative methods of restoring the displaced organ may be employed. Of earlier methods, the first operator, Duret, fixed the stomach to the parietes, but this is objectionable as limiting the normal stomach movements. Better results can be obtained by raising and fixing a dropped liver, if present, and by shortening the gastro-hepatic omentum by pleating. This operation is often most satisfactory in its results, as I have proved on several occasions, and is much more likely to cure than the less simple procedure of gastro-enterostomy. In association with the plication of the omentum, adhesions holding up the pylorus to the under-surface of the liver are divided, so that while the rest of the organ is raised the pylorus is dropped and the whole organ given a useful slope from left to right. Gall-stone trouble, if present, can be treated, and the right kidney fixed, by an anterior nephropexy.

Small intestines.—Undoubtedly the dropping of the coils of the small intestine occurs with the falling of the other organs, and it is particularly causative of fulness of the lower and flattening of the upper abdomen. Such falling, I believe, has a secondary effect in displacing pelvic viscera. I have had no experience of the direct operative treatment of this condition, but obviously a well-fitting belt will do much good.

Large intestine.—Frequently when the stomach is empty the transverse colon is not transverse, and does not sag down, but is convex upwards, lying in front of the stomach. This can be quite usually

noted upon opening the abdomen in the middle line above the umbilicus, the transverse colon being the first viscus reached.

According to Glenard it is the displacement of the hepatic flexure of the large intestine which is the first step in the series of phenomena resulting in general proptosis. With the liver and stomach, or with the stomach alone, the transverse colon necessarily becomes displaced, and if the colon thus comes down in the middle while it is held up at the two flexures stagnation and decomposition of its contents, producing the various kinds of colitis, are very likely to ensue. Here we come to another class of case, the colitis class, where the non-operative treatment, as described by Hale White, consists of rest, abdominal massage, and the administration of castor oil, and the operation of appendicostomy, or ileo-sigmoidostomy or excision of the large bowel. The non-operative treatment which cures the stomach dropping will often cure the colon dropping too. Or, when the small omentum is plicated the gastro-colic omentum may be plicated also. Of "removal of the large bowel" for chronic constipation, as advocated and practised by Arbuthnot Lane,* I have had no experience. Mr. Lane gives but the briefest account of his cases, and many are recorded at so short a date after operation as to give no proof of the permanency of the cure. Then, patients of this class are highly neurasthenic, and might possibly be relieved temporarily by suggestion if you merely opened the abdomen and stirred up the contents with the finger. They require watching for many years before being regarded as permanent cures. Possibly in a very few cases this severe operation (of which the mortality in the series of cases published by Lane is at least 18 per cent.†) may be advisable when "auto-intoxication" is very severe, and it is impracticable to resort to other measures of relief. Of cases in my own experience I will refer to a case of anterior transplantation of the sigmoid. In this case a neurotic man only had an action of the bowels once weekly. On opening the abdomen the sigmoid flexure was found short, straight, and tightly fixed by strong external bands in the iliac fossa and pelvis in such a way as to be sharply angulated with the descending colon. It was released, the angle straightened, and the bowel fixed to the parietes much more anteriorly. Relief of pain followed, the action of the bowels became more normal, and the man returned to work. The improvement was maintained six months later.

Rectum.—For severe rectal prolapse in a child I have performed the operation of sigmoidopexy. The abdomen is opened, and, the prolapse being reduced by an assistant, the bowel is pulled taut and the sigmoid anchored to the peritoneum of the lateral abdominal wall. The cure obtained in my case has been permanent for nearly a year.

Adhesions.—I should like to say a word or two about adhesions. I think surgeons are too ready to assume that adhesions will form in the abdomen without any very definite cause, *e.g.* adhesions in connection with the large intestine are looked upon as the starting-point of its disturbances. But adhesions themselves must have a cause. Normal peritoneal surfaces, *e.g.* the upper surface of the liver and the under-surface of the diaphragm, are in contact with one another and rubbing against one another all life long, and still there are no adhesions between them. Adhesions are the result of inflammation. This, in the case of the hollow abdominal viscera, spreads from the inside to the outside through the wall of the organ and affects the peritoneum. The infection then reaches by contiguity the adjacent peritoneum and the two surfaces adhere. Or an irritant, infective or otherwise, is introduced into the peritoneal cavity and similarly produces adhesions of the peritoneum it reaches. There is much yet to be learnt concerning the patholog of adhesions. The undoubted self-freeing of adhesions when their cause has subsided is a matter of much interest. This, however, is undoubted, that adhesions cannot form without a definite cause.

Glenard's disease.—The treatment of this or general visceral proptosis I have spoken of under the headings of the various viscera. General medicinal and hygienic treatment, the rest cure, massage and a proper belt will give great relief to the majority of cases. When these fail or are inapplicable, or where—as is often the case—organic disease is suspected, the abdomen is opened in the median line, above the umbilicus, the incision being lengthened downwards as far as necessary. The median incision is particularly preferable, as it does not interfere with already weakened muscles. Liver, stomach, and colon are raised and fixed as already

* 'Brit. Med. Journ.,' vol. i, 1908.

† *Ibid.*

mentioned, and one or more of the following further procedures may be called for: Operation on the gall-bladder, freeing pyloric adhesions, freeing omental adhesions, straightening the jejeunum at the duodeno-jejunal flexure, altering the position of the sigmoid flexure, anterior nephropexy, appendicostomy, and finally hysteropexy by pleating the round ligaments—probably the best method of fixing a purely displaced uterus.

Presence of organic disease.—Operations for displaced viscera often bring to light the unsuspected presence of organic disease. Cancer, ulcer and tubercle may each be met with. Where, in spite of other treatment, there is *progressive loss of weight*, this is always to my mind a very good reason for opening the abdomen.

I must apologise for these somewhat fragmentary remarks. They are based upon some thirty cases I have dealt with surgically, and upon others in which I have advocated milder measures. I feel that it is a subject interesting to all sections of the profession, and upon which all can throw some light, and bearing that in mind I have ventured to bring it before you to-day.

February 8th, 1909.

Morris's Point of Tenderness as an Aid in Diagnosis.—J. C. Hubbard declares that the point has by no means the importance attached to it by Morris. The abdominal lymphatic glands, becoming secondarily inflamed, cause tender areas. The area found tender on examination is somewhat of a guide to the organ primarily infected, and therefore in certain cases may be an aid in diagnosis. In acute appendicitis tenderness at Morris's point is of less importance than the symptoms caused by the appendix itself. In chronic appendicitis tenderness at the point may be of distinct diagnostic value. Tenderness at this point, even though the only physical sign, makes the diagnosis by the rule of chance probable. When combined with tenderness at McBurney's point the diagnosis becomes more certain. Its absence does not rule out appendicitis, and it may occur in other conditions.—*Medical Record*, vol. lxxiii, No. 3.

WITH MR. GORDON WATSON IN THE WARDS OF THE METROPOLITAN HOSPITAL.

THREE CASES OF LAPAROTOMY FOR ACUTE PERFORATION.

1. A Case of Perforation in Enteric Fever: Laparotomy; Recovery.

F. M—, a girl, æt. 11 years, was admitted to the Metropolitan Hospital under the care of Dr. Cautley on the fifth day of an attack of enteric fever.

The disease ran a straightforward course for three weeks after admission. The temperature was never below 103° F. at night, on two nights it went up to 104° F., and twice to 104·6° F.

The general condition was fairly good. There was no hæmorrhage and no sloughs were passed. There was an average of two stools a day. The mental condition was semi-conscious.

At 2.10 p.m. on the *twenty-sixth day* of the disease the child suddenly cried out, and was seen to lie with her knees drawn up and her hands placed over her abdomen. Her expression was anxious; respirations were shallow and rapid; the pupils dilated, and the pulse small and increasing in frequency. The abdomen showed general distension (slight distension had been noted during the past twenty-four hours), and was rigid and tender all over; there was only slight movement on respiration. On percussion there was marked dulness in the flanks and the liver dulness was diminished.

At 2.30 p.m. a leucocyte count was made and found to be 16,000. *Ten days previously the count had been* 6000. The blood-pressure was 90 (systolic). *The liver dulness was now completely lost.*

At 2.30 p.m. I saw the patient in consultation with Dr. Leonard Williams and decided on immediate operation.

The patient was taken to the theatre, after a hurried preparation of the abdomen. At 3.16 the operation was commenced. A 3-inch median incision was made below the umbilicus. On opening the abdomen gas and fluid containing intestinal contents at once escaped. A perforation was

quickly found about eighteen inches from the ileo-cæcal valve, and closed by four Lembert sutures inserted transversely to the long axis of the bowel. A *second ulcer* rather below this was found to be *on the verge of perforation* and stitched over in the same way. At the ileo-cæcal valve a small oozing point was found (? ruptured adhesion) and secured by a stitch. The peritoneum everywhere was slightly injected, but quite glossy. Two incisions were then made, one in either flank, large drainage-tubes inserted, and the peritoneal cavity flushed with hot saline solution. The median incision was closed with one layer of silkworm-gut sutures, and the sutured portion of intestine allowed to lie subjacent to the wound. The operation was completed at 3.37—in twenty-one minutes.

Condition after the operation.—The general condition of the patient at once improved. The temperature fell to 98·4° F., and the patient fell into a natural sleep. On the following day the patient became conscious, the expression was placid, and the typhoid condition had passed off. For four days after the operation the temperature did not rise above 99° F., but on the fifth day there was a rise to 100·4° F., and the median wound began to suppurate. The stitches were removed, the wound gaped, and a fæcal fistula ultimately formed. Except for the fistula the child rapidly recovered, and was sent to a convalescent home four months after operation with a small sinus, but no fæcal extravasation. Three months later the patient was re-admitted fat and well. The sinus was then closed by dissecting away the mucous lining, without opening the general peritoneal cavity.

The child is now, twelve months after the first admission, in perfect health, and the fæcal fistula has remained closed, though there is still a small superficial sinus.

The successful result which followed operation in this case may be attributed to an immediate diagnosis followed by operation an hour after the first symptom of perforation. Additional factors to recovery were, the age and general condition of the patient, the short duration of the operation, the search for other ulcers about to perforate, and the suturing over of one of these.

The high mortality which has followed operation for perforation in enteric fever must be attributed either to the shock of the operation on a patient already greatly exhausted and in a highly toxic condition, or to peritonitis having become general before laparotomy was performed, or to a combination of these two.

The circumstances surrounding the case under consideration were such as to eliminate these factors, and a very noticeable feature in the case was the marked improvement in the typhoid condition which followed operation.

When put back to bed the child fell into a natural sleep for the first time (since admission), and on awaking was rational instead of typhoid; some colour returned to the face; there was a desire for food. The temperature fell and continued almost on normal lines (until suppuration occurred); the acute toxic condition had passed off with the perforation and its treatment.

This, I think, may be largely attributed to the washing out of a very large quantity of organisms and toxic material, which had passed from the intestine into the peritoneal cavity, and to the substitution of a large quantity of hot saline, which was, doubtless, rapidly absorbed by the peritoneum.

A *diagnosis* will not often be as easy as it was in this case, where all the classical symptoms of acute intestinal perforation were present.

Several other conditions may simulate perforation. Perforation may occur so secretly that only the onset of a subsequent acute general peritonitis may attract attention by its acute symptoms. It is, therefore, very necessary to be not only familiar with these other conditions, but to watch most carefully for pre-perforative symptoms.

I do not propose to discuss the differential diagnosis when perforation is suspected. Apart from intestinal hæmorrhage, which may closely simulate a perforation, the following conditions have been met with in enteric fever, for which laparotomy has been performed on the assumption that a perforation of the intestine has occurred: iliac thrombosis, ruptured mesenteric gland, pneumonia with involvement of the diaphragmatic pleura, cholecystitis, and ruptured adhesions. Cases have been recorded of peritonitis occurring without visible perforation, and have been confirmed post-mortem. One such case was under my care in the hospital in 1905 (*vide* 'Lancet,' May 6th, 1905). A boy, æt. 9 years, was admitted with acute general peritonitis; appendicitis was suspected to be the

cause. I explored the abdomen and found general purulent peritonitis; the pus was thin and odourless, and there was *no* free gas. Post-mortem the case proved to be one of enteric fever in the second week. Very careful examination revealed no evidence of past or present perforation.

Pure cultures of the *Bacillus typhosus* were obtained from the spleen and heart's blood. That no perforation had occurred in this case seemed evident from the *condition of the ulcers, the absence of free gas, fæcal odour or extravasation.*

Without doubt cases occur in which ulceration approaches so near the peritoneal coat that organisms can pass through and give rise to peritonitis. In a report of a committee of the Boston Society for Medical Improvement,* on twenty-four cases operated on for symptoms of peritoneal infection in typhoid fever, two cases were shown to be due to "pre-perforative" necrotic areas of the intestinal wall, without actual perforation.

These cases serve to emphasise the importance of recognising the "pre-perforative" or "pre-extravasation" stage. In the twenty-four cases alluded to above the diagnosis was apparently made *later* than it might have been in fourteen, in three a mistaken diagnosis was made, and *only in seven* was an *immediate* diagnosis made.

From the study of recorded cases it is evident that more cases of perforation present symptoms of a gradual than of a sudden fulminating onset. This is a clinical fact of the utmost importance to the surgeon, because when the onset is gradual acute symptoms do not appear until *general peritonitis* is established.

We must recognise that the perforative cases fall into two main groups, the "*fulminating*" and the "*leaking*," just as they do in appendicitis. There may be a stage of local peritoneal infection, with or without adhesions, which are not often met with. This local infection may cause general infection without actual perforation. *Local infection of the peritoneum causes pain of greater or less severity.* In mild cases of fever this pain may be as prominent a feature as in appendicitis before perforation; in severe "typhoid" cases it may not be complained of, thus rendering an early diagnosis most difficult. When acute symptoms do occur in this latter class it generally means that acute general peritonitis has already occurred; on the other hand, sudden acute symptoms in mild cases generally mean sudden perforation with extravasation. There seems to be no relation between the severity of the disease and the likelihood of perforation.

Intestinal hæmorrhage must be regarded as a danger signal of perforation; it indicates extensive ulceration and close proximity to the peritoneal coat, and not infrequently precedes a perforation for a day or two beforehand.

It cannot be too strongly urged that a *complaint of abdominal pain*, especially if localised and accompanied by tenderness and spasm, is not a frequent occurrence in enteric fever *unless there is some peritoneal infection*, local or general. This is more especially true in the mild cases.

If peritoneal infection is suspected much help may be gained by studying the leucocytes. During the course of the fever there is usually leucopenia, and if with the onset of pain there also occurs a gradually rising leucocytosis, the suspicion of peritoneal infection receives strong confirmation.

If a white blood-count is to be of any diagnostic value in the presence of abdominal symptoms, it is essential that previous counts should have been made. A single count may be misleading. On the one hand a very moderate count, such as 10,000, may in a case of enteric fever represent a definite leucocytosis, which can only be shown by comparison with previous counts; and on the other the leucocytosis which usually occurs with peritoneal infection is often very transitory, and may vanish when general peritonitis is established if the patient is too exhausted to react against the infection. If the process remains localised or local suppuration occurs, there is ordinarily a persistent leucocytosis.

The question of the anæsthetic is important. In the case under consideration very little chloroform was necessary, and it was administered with great skill, and not followed by any vomiting. In future, should I be called upon to operate for perforation in enteric fever in an adult I should be inclined to make use of spinal anæsthesia, especially if there was any pre-existing bronchitis or marked collapse. My limited experience of spinal anæsthesia has been most favourable. It has proved a most valuable method in cases of gastric perforation. At the Johns Hopkins Hospital many

* By Shattuck, Collins Warren and Farrar Cobb, 'Boston Medical Journal,' June 28th, 1900.

operations in typhoid cases have been performed under *local* anæsthesia by the infiltration method. Harvey Cushing (in the 'Philadelphia Medical Journal,' March 3rd, 1900), very strongly advocates this method, and gives a detailed account of five cases of enteric perforation (or suspected perforation) operated on under local anæsthesia. Without doubt the risk of the operation is diminished, and for this reason local anæsthesia should be the means of securing more frequent explorations in suspected cases of perforation.

2. A Case of Perforation of a Gastric Ulcer, which Occurred in an Apparently Healthy Girl while at Work in the Hospital; Laparotomy; Recovery.

A ward-maid, æt. 21 years, was suddenly seized with acute abdominal pain while drinking a cup of tea at 4.30 p.m. *She had been at work as usual and in her ordinary health.* She had occasionally suffered from indigestion, but had never had symptoms suggestive of gastric ulcer, and had been taking ordinary food up to the time of the attack of pain. When seen a few minutes after the attack of pain in her room she was lying on the bed with both legs drawn up, and in acute pain referred to the pit of the stomach. The face was flushed and the forehead wet with perspiration; the tongue was slightly furred, the pulse 86 and the temperature 97·4° F.; *she had not vomited and did not feel sick.* The abdominal muscles were exceedingly rigid, movement was diminished, and tenderness most marked over the right rectus in the region of the pylorus. The liver dulness commenced at the sixth rib and extended to the costal margin; there was no dulness in the flanks or in the right iliac fossa and the abdomen was resonant all over. Nothing abnormal was felt *per rectum.*

She was removed to the ward and hot fomentations applied to the abdomen. One hour later her condition was unchanged except that there was some diminution of liver dulness. There had been no vomiting and no increase in the pulse-rate; the pain was unrelieved by the fomentations. A leucocyte count was made and the result was 15,000 leucocytes to the c.mm. The patient was seen at intervals and presented much the same signs and symptoms, except that the pain was a little easier. At 9.30, five hours after the onset, I decided to explore the abdomen for perforated gastric ulcer. *The liver dulness had then completely disappeared* and the abdomen was hyper-resonant and fixed; the pulse-rate was 96, the temperature still subnormal and vomiting absent. On opening the abdomen free gas and fluid escaped. A perforation was found close to the pylorus near the lesser curvature and sutured. The abdominal cavity was flushed out with saline and drainage tubes put into the right flank and above the pubes. The operation lasted twenty minutes. The patient was kept in the Fowler position and made an uninterrupted recovery, and was sent to a convalescent home eighteen days after the operation.

Remarks.—Several points of interest may be noted in this case.

(1) The patient was watched practically from the very moment of perforation. (2) Perforation was the first indication of the presence of an ulcer in the stomach. (3) In spite of the sudden and acute invasion of the peritoneum no vomiting occurred, and the pulse-rate never reached 100. (4) The liver dulness was observed to gradually disappear.

The loss of liver dulness is a sign of perforation of a viscus which has received great attention in the text-books, and is by no means always met with, though more often in gastric perforations than in intestinal. In perforation of the small intestine, if loss of liver dulness is waited for, it means waiting for the onset of general peritonitis. In this case in the absence of vomiting, previous history of illness or increased pulse-rate, *the loss of liver dulness rendered the diagnosis practically certain*, and enabled colic to be excluded.

It will be noticed that loss of liver dulness was also noticed in the case of enteric perforation, though the diagnosis would have been practically certain without it.

3. A Case of Strangulated Femoral Hernia (Richter's) Reduced by Laparotomy; Subsequent Perforation, Laparotomy, and Death.

A woman, æt. 30 years, was sent up to the hospital with a diagnosis of appendicitis and peritonitis. On admission she was extremely collapsed, with a rapid running pulse and subnormal temperature. Her pupils were pin-point; she was very drowsy, and could with difficulty be got to answer questions. The history of her condition on admis-

sion was briefly this: One week ago she took some liquorice powder for constipation, with no result. Five days ago she took a large dose of salts and the bowels acted. This was immediately followed by severe abdominal pain and sickness, for which she consulted a doctor. Since that time she had been acutely ill, sick after everything, bowels not opened, general abdominal pain, more marked in right iliac fossa.

On examination the abdomen was found to be a little distended, but not markedly so. Nothing abnormal could be felt in the right iliac fossa or elsewhere in the abdomen, or in the pelvis by bimanual examination. There was general abdominal tenderness, but no definite local tenderness or other signs. She was evidently so much under the influence of morphia that her symptoms were considerably masked, and pain was not a prominent feature. The hernial orifices were examined and *nil* found.

It was evident that she was acutely ill with some abdominal complaint, that she was collapsed from vomiting, and that her symptoms were masked by morphia. The most likely diagnosis seemed to be internal strangulation or gangrenous appendicitis, with peritonitis and a subnormal temperature due to collapse. Immediate operation was decided on, and I opened the abdomen in the right iliac region through the sheath of the rectus. The appendix was at once seen to be normal. Further examination revealed a coil of small intestine running down to the right femoral ring and there caught up. Gentle traction easily reduced this, and it was then seen that there had been a strangulated partial enterocele in the femoral canal. Only a portion of the lumen of the gut had been nipped. The strangulated portion when examined was congested and a little discoloured, but appeared in quite good enough condition to recover. The gut immediately above was somewhat distended and congested, and the vessels in the mesentery also congested. The condition of the patient was so bad during the operation that resection would have been out of the question, nor did I think that the condition of the gut required it. Had I thought otherwise I should have been content to make an artificial anus and tie in a Paul's tube. The gut was returned and the wound closed. The patient was so collapsed that strychnine, brandy, and coffee enema and oxygen were resorted to before she could be sent back to bed. For the next few hours her condition was extremely critical, and then she gradually improved, and when seen by me ten hours after the operation she expressed herself as very comfortable. Her bowels had acted three times, her abdomen was not distended, she had no pain, and her pulse was good. She was taking small doses of milk and water by the mouth, and had only once been sick, soon after the operation. She seemed to have recovered from her strangulation. Two hours later she complained of sudden abdominal pain and vomited. This was followed by a rapid increase in the pulse-rate and abdominal distension. When seen by me three hours later she was at death's door, with all the signs of intestinal perforation and general peritonitis. *The liver dulness was completely absent.* Operation seemed out of the question. Intra-venous infusion of saline (three pints) was at once carried out, and then with the addition of brandy and strychnine she rallied snfficiently to enable me to reopen the wound and to find, as I suspected, that the gut had perforated at the line of constriction. The perforated portion was brought up to the wound and a tube tied in. A large supra-pubic drainage-tube was also inserted and the cavity washed out with hot saline. Every effort was made to combat the shock, but the patient died two hours later.

Remarks.—This case emphasises the extreme danger of returning strangulated gut in cases of femoral hernia, even though not gangrenous.

It adds one more to the list of undiagnosed Richter's herniæ.

It condemns morphia in acute abdominal complaints.

It is a noticeable feature that in this case, as in the two other cases, free gas in the peritoneal cavity was recognised by the loss of liver dulness *quite early* after the onset of perforation.

February 8th, 1909.

Cervical Ribs.—Schaefer says that cervical ribs are not at all so rare as has been thought previously; 139 cases have been described before 1894, and the use of X rays since that date has shown that in many cases the presence of this anomaly has escaped attention. He has observed three cases of cervical ribs. The first was a woman whose affection was diagnosed as incipient pulmonary tuberculosis.—*Medical Record*, vol. lxxiii, No. 3.

INTERNATIONAL CLINICS. Eighteenth Series, vols. i and ii. (J. B. LIPPINCOTT Co., Philadelphia and London, 1908.)

ABOUT a half of the first of these volumes is occupied by a synopsis of the progress of medicine and surgery during the year 1907. The greater part is a brief summary of papers, and will prove of little use to anyone who does not consult the originals, while it is distinctly out of keeping with the rest of the volumes. We must note, however, that the surgical section, by Dr. Joseph C. Bloodgood, is written in a different manner and gives a full account of recent advances in the diagnosis and treatment of surgical affections. In this section also there are numerous illustrations, plain and coloured, showing the clinical and pathological appearances of morbid growths, which are well selected and well executed.

Amongst the clinical papers there is one by Dr. R. D. Rudolf, who tries to shake one of our most cherished beliefs that the average temperature of the human body is 98·4° F. During the winter months he found that over 70 per cent. of the surgical cases in the General Hospital, Toronto, showed an average temperature well below 98·4° F., while in the summer months the percentage fell to about forty. His investigations have led him to place the average temperature of a healthy individual at rest in a temperate environment between 97·2° and 98·4° F.

In the second volume Dr. Theodore Diller discusses the subject of pain as the chief or sole expression of a psychic state. His remarks on this familiar subject and his illustrative cases are well worth reading. The patient who goes about with a pain and a long story, a pain which defies the drugs of the physician and the knife of the surgeon, is a veritable object of compassion, but perhaps his real purpose in life is to replenish the coffers of many doctors. American writers are said to be ambitious of elaborating a new language, and from this volume we should judge that they are well on the way to success. Expressions like the following meet one on every page: "He was loaded down with salicylates"; "a spot of tenderness the size of a silver quarter"; "the patient is a robust, stocky-looking boy." Fortunately there is enough of King's English in the context to enable one to grasp the meaning, so that the only effect so far is to add a certain brightness, if not elegance of style to the clinical lecture. Dr. Hallopeau, of Paris, writes hopefully of the addition of atoxyl to the list of remedial measures in syphilis. The symptoms of poisoning which have been recorded in several cases are, he thinks, the results of over-dosage, and may be avoided by giving first at intervals of two days two injections of 0·75 grm. of atoxyl, and then four injections of 0·50 grm. separated by an interval of three days each. As usual these volumes contain contributions from Europe as well as from America, and not only are medicine and surgery included, but also all the various specialties. Each volume forms a valuable addition to the practitioner's library.

Serum Treatment of Thyroidism.—Rogers and Beebe describe their work as the first attempt to treat disease in the human subject by means of a specific cytotoxic serum, and their conclusions, subject to revision as experience increases, are as follows: The serum has a specific effect in neutralising the toxic action of the thyroid secretion; as a therapeutic agent it gives results which cannot, in many cases, be attained by any other medical means; not all cases presenting symptoms of thyroidism can be treated successfully with serum, because not all cases are purely hypertrophic in origin. The following types are favourable for serum treatment: Typical exophthalmic goitre in the early stages, including the incipient, the mild, the severe, and those extremely severe forms which develop very rapidly and have been described as the acute toxæmic type, resembling malignant endocarditis: Typical exophthalmic goitre in cases which may have existed for some time in subacute form with occasional exacerbations, but without marked secondary changes. The rapid amelioration of symptoms in the acute toxic cases, similar in most respects to the well-accepted instances of neutralisation of toxin by antitoxin, is a weighty argument in favour of believing the symptoms to be due to the toxic effects of hyperthyroidism. The beneficial results of combined treatment, especially in the older cases, indicates a dysthyroidism as well as hyperthyroidism as a factor in the production of symptoms.—*Journ. A. M. A.*, vol. lii, No. 3.

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A CLINICAL LECTURE

ON

TROPHO-NEUROSES AND ANGIO-NEUROSES OF THE EXTREMITIES.

AMYOTROPHY, SYRINGOMYELIA, HERPES, RAYNAUD'S DISEASE, COLD HANDS AND FEET, ERYTHROMELALGIA, HYPERIDROSIS, URTICARIA HYPERKERATOSIS, AND ANOMALOUS ŒDEMA.

Delivered at the West-End Hospital for Diseases of the Nervous System, Welbeck Street, W.

By THOMAS D. SAVILL, M.D.Lond.,
Physician to the Hospital.

GENTLEMEN,—I happen to have a series of cases at this and at my skin clinique illustrating symptoms which are referable to the ends of the extremities, and I have thought that it might be interesting to collect and demonstrate them to you this afternoon.

At the outset I would like to remind you that the ends of the extremities—that is to say, the hands and feet—are exposed in common to certain conditions of environment. Both hands, for instance, are exposed to the air, to the action of of water, and to occupations. And these environments may give rise to eruptions which are more or less symmetrical. It is well not to forget this identity of local conditions, and not to mistake this for a symmetry due to spinal, circulatory, or toxic causes. But I wish more particularly to draw your attention to two special physiological peculiarities which the ends of the extremities present in common. First, they have longer neurons than other parts of the body, and consequently they show a special tendency to trophic changes, because it is the end of a neuron furthest from the ganglion-cell which first shows nutritive changes. The second peculiarity, which, in my opinion, is even more important, is that in each

hand and foot there is what might be called a "dead end" to the circulation, a kind of back-water where the blood-stream is sluggish. This is, to some extent, a mechanical or dynamic factor, which in health is compensated for largely by the vaso-motor mechanism. But it is here where vaso-motor defects are so apt to show themselves. This question of the circulatory dead end is also important because in certain toxic states the toxins are delayed longer at the ends of the extremities, and therefore have more effect than in positions where the blood flows more freely. Bearing these two common peculiarities in mind we see that there are two groups of cases that we shall have to consider which correspond with them—the *tropho-neuroses* and the *angio-neuroses*, both of which are specially liable to affect the ends of the extremities.

True Tropho-neuroses.

First, then, we meet with nerve cases, tropho-neuroses, such as lesions of the lower motor neuron. This patient, a man, æt. 60 years, is a well-marked example of progressive muscular atrophy, chronic anterior poliomyelitis. The story is that five or six years ago he *first began to notice that his hands were wasting*. Now, as you see, he has marked wasting of the small muscles of the hands. The interossei and the thenar and hypo-thenar muscles are chiefly affected, and he is already beginning to get his forearms involved. By-and-bye the muscles of the shoulder girdle will be affected. The main lesion is one of the anterior motor cells (chronic anterior poliomyelitis), and you have here a fairly perfect type of lower neuron paralysis, of which the essentials are flaccid paralysis, muscular wasting, and electrical changes (changes which are not always marked in chronic forms of this disease), with loss of the tendon reflexes of the particular muscle involved. These are the four characteristics of a lower neuron lesion as against those of an upper motor neuron lesion, in which you get muscular stiffness, increased tendon reflexes, no wasting (excepting that of disuse), and no electrical changes.

There is another nerve disease, in which the lower sensory neurons are affected as well as the motor neurons, but which sometimes resembles the foregoing in its onset. That disease is syringo-myelia; and in these transverse microscopic sections of the cord which I show you the changes which occur are well shown. You will notice, even with the naked eye, that there is a large increase in the size of the central canal; in places a space nipped off from the central canal appears; there is cavitation, in fact, with an overgrowth of gliomatous tissue in the walls of the cavities. This is the essential lesion in this disease, a progressive gliomatous cavitation. Whether the cavitation is congenital or not is a point upon which pathologists are not as yet agreed. But the gliomatosis is probably secondary, and when gliomatosis occurs we get four sets of symptoms. (1) Quite early we meet with sensory changes, and among them there is that characteristic feature of the disease first pointed out by Charcot—loss of thermal sensation. The explanation of this is that the thermal tracts lie close to the central canal, and are early involved in the gliomatous overgrowth. But (2) the anterior horns are also involved, and muscular wasting, starting usually in the hands, results. (3) Subsequently the posterior horns and ganglia become involved by pressure or gliomatosis, and many different trophic changes, referable mainly to the hands or feet, occur in the nails, skin, and tissues. Painless whitlows result (these were formally called Morvan's disease, but are now regarded as symptoms of syringomyelia). The patient from whom these microscopic sections were taken after death, shed portions of the skin of her hands, which slipped off like the finger of a glove. (4) Pressure by the gliomatosis on the lateral columns occurs, and that gives rise to muscular rigidity. This patient, a woman, æt. 50 years, first came to this hospital in October, 1907, with a history for three years of pain in the right side of the head and numbness of the right upper extremity and partial loss of power in that hand. On examining her we found that there was slight wasting of the thenar and hypo-thenar muscles in the right hand, and thermal anæsthesia. This still exists in a notable degree, for although the common sensation is little, if at all, diminished, she is not able, with her eyes shut, to tell the difference between the hot and cold test-tubes placed on the skin of her limb. Incidentally, I may mention that she also exhibits another feature of interest in that she has some of the signs of paralysis of the right cervical sympathetic. The chief signs of sympathetic paralysis are four: First, contraction of the pupil on the affected side; secondly, narrow-

ing of the palpebral fissure; thirdly, recession of the eyeball, or enophthalmos. This patient has not got the pupil sign, but she shows the palpebral and eyeball sign, and she has, in addition, the fourth sign, that is to say, absence of sweating, or anidrosis on the affected side. She also complains that this side does not flush when the other does. These symptoms, I may mention, were at first regarded as an independent lesion of the sympathetic, but it is not uncommon to find such symptoms in syringomyelia. They appear to be due to involvement of the pupil dilator fibres within the cord.

These, then, are two instances of neurotrophic lesions. In both, the ends of the extremities are first affected, and in both it is the trophic or nutritional nerve-fibres derived directly from the spinal cord which are involved, resulting, in the first case, in muscular wasting, and in the second in motor and sensory symptoms. "Perforating ulcers" on the feet and elsewhere are apt to occur in association with tabes, and it is probable that these also belong to the same group.

Ridges in the nails, arising during a severe illness, are often mentioned as instances of true trophic change, but for myself, I cannot see why they should not be due to a defect in the vaso-motor apparatus of the limb, which undoubtedly regulates the supply of nutrient fluid (*i. e.* the blood) to the limb.

Herpes and herpetic vesicles should, I believe, be classed as due to genuine lesions, either of the true trophic fibres in the cerebro-spinal nerves, or of the trophic centres in the spinal cord. Herpetic vesicles on the fingers were one of the changes which occurred in the hands of a case of fibroma of the median nerve, which arose after division of that nerve, which I published in 'Brain' some years ago.* Herpes is now known to arise from injury or disease of the posterior root ganglia. I believe that herpes on the hands and feet are much more frequent than is generally recognised; the vesicles break, being very liable to injury in that situation, and thereby the typical appearances of the group of herpetic vesicles on a crimson base are destroyed. I believe that quite a number of cases which physicians, even men of experience, regard, from the appearances seen at the time, as cases of patchy eczema on the hands and feet, are in reality herpes in their origin. Look, for instance, at this girl, æt. 16 years. You would think at the present moment that she had patches of weeping eczema on her hands. But look at this sketch of her hands, which was made a couple of weeks ago. There are five typical herpetic groups on the two hands. Her history is also interesting. She had her first attack at the age of one year, her second at the age of thirteen, her third at the age of fourteen, and her fourth started a month ago, first with itching, and then with herpes, the history in each case being typically that of herpes zoster.

* 'Brain,' 1893, p. 452.

Angio-neurosis.

We now turn to the second group of cases, all of which I believe to be angio-neurotic or vaso-motor in origin. There are some eight clinical conditions, and they are all, in my belief, due to faults in the vaso-motor apparatus, coupled sometimes with toxæmia. They are mainly skin conditions, and most of the patients are from my clinique at St. John's Hospital for Diseases of the Skin.

The first is the classical condition known as Raynaud's disease, in which the changes in the skin may lead to ultimate gangrene. A case of this I am unable to show you. Secondly, we meet with a number of conditions which may all be classed as being due to general feebleness of the circulation: Cold hands, blue hands, dead hands, and such like conditions, which are so common that I need not show them to you. They may often be improved by Blaud's pills. Thirdly, we have a condition which was called erythromelalgia by Weir-Mitchell. This is a painful red swelling of the hands and feet. This patient, Mrs. T—, æt. 61 years, is a very good example of the condition; her case was fully described in one of my previous clinical lectures.* She came complaining of pain, swelling and redness in the hands, dating from four years previously. The condition was, in 1897, a most distressing one when I first saw her. She got very much worse whenever she went to bed, or even when she lay down horizontally on the sofa. This prevented her having any rest. The treatment which did her most good was galvanism, the constant current frequently applied. She was also given calcium chloride and other remedies, and ultimately she got almost quite well. There is still some redness of the hands and feet, but she comes now with erythematous patches on

* 'The Lancet,' June 1st, 1901.

the feet, which somewhat resemble erythema nodosum, from which she has suffered from time to time. These red patches and the erythromelalgia are, I believe, a toxo-angio-neurotic lesion.

A fourth condition is hyperidrosis — excessive sweating of the hands and feet. This girl, Edith S—, æt. 21 years, a typist, has suffered for three or four years, always worse in the summer, from this condition. The hands sweat so much that it is difficult for her to work her machine, and when they are at their worst she gets miliary vesicles appearing beneath the thick epidermis of the palms, such as one finds in association with excessive sweating anywhere. And with these two symptoms I would ask you particularly to notice there is a redness, a flushing of the fingers, which is always worse when the other symptoms are worse, the redness being most marked at the ends of the fingers. Ergot and adrenalin have done her good, and enabled her to do her work, but when she leaves them off she gets bad again. Locally, the most efficacious remedy for these cases is: Salicylic acid 10 gr., oxide of zinc 1 dr., starch 1 dr., lard 1 oz.

The fifth case I have to show you, that of Emily S—, æt. 43 years, exhibits a curious condition of concavity of the nails, which has come on gradually during the last eight or nine years. I admit that it is an open question whether these are a true tropho-neurosis or whether the condition is an angio-neurosis, but in view of the fact that from time to time she has exhibited various flushings, sweatings, and tiny miliary vesicles beneath the epidermis of the palms of the hands, during the same period of time—thirteen years—I suggest that the nail lesions may be of the same angio-neurotic origin as the other symptoms.

The next case is a very old patient of mine, named Mary H—, æt. 28 years, a housemaid. She first came to me eight years ago because her face and nose became swollen when she blew her nose, so that she could not answer the door; and because she got swelling of her hands, also of an urticarial nature, whenever she handled the broom. In short, at that time she had to leave her situation because of this recurrent urticaria arising whenever any pressure was exerted anywhere on the skin. She got well, though I forget for the moment what were the remedies which she took. Now she comes complaining of a recurrence of this urticaria-like swelling in the hands alone. It varies from time to time, and she says she is always feeling tired and gets twitchings of the limbs at night. In short, I think that she has a tendency to urticaria of the hands only this time, and the disease is mainly a chronic toxæmia.

There is one more lesion—hyperkeratosis—which I can illustrate to-day. The patient, Amy P—, is a very interesting case. You see she has horny thickening of the palms of both hands, quite symmetrical. It is not very great, but it makes the palms of the hands very hard and rough, and from time to time they get sore. I may note, in passing, that the entire surface of the body has a somewhat ichthyotic tendency, not very marked, but I think it is sufficient to make the angio-neurotic disturbance in her take the form of hyperkeratosis palmaris. She gets the same flushing and alternating pallor that the others had. In some cases hyperkeratosis is an acquired condition, coming on in childhood or adult life, but in some it is congenital, as in this case. And not only so, but it is here apparently hereditary, or at any rate familial. There are eight sons and daughters in this generation, and three of these eight have a similar condition on the palms of the hands and soles of the feet.

The *œdème bleu* and *œdème blanc* of Charcot always, as far as my memory serves me, affect the ends of the extremities; the hands, wrists or forearms, or in rare instances, the feet. This consists of a localised spherical œdematous swelling occurring in hysterical subjects, and in association with redness or pallor of the skin of the extremity. There are other conditions of anomalous œdema of this character, all somewhat rare, which have been described by various authors. The subject is a somewhat abstruse one, and I must not stay to discuss this condition now (a note has since been added below). But the history and concomitant symptoms of these cases appear to me to be explicable only upon an angio-neurotic hypothesis. There is, in some people, undoubtedly, an instability of the local peripheral vaso-motor mechanism, and this derangement of the vaso-motor mechanism is manifested by an œdema of the locality. This œdema must be, in my judgment, due to a constriction or a dilatation of the arteries, lymphatics, or veins going to or from the part. Nothing else is capable of producing this œdematous swelling. They do not always pit very

deeply on pressure, but the fluid can generally be expressed from the part to some extent.

In conclusion, gentlemen, it appears to me that there are always three factors to be remembered in considering cases like those which you have just seen in what I call my angio-neurotic group. First, there is the sluggishness of the circulation, and the fact that you have to deal with a dead end, a partial stasis. This is more or less a *mechanical* factor. Such stasis leads more readily to local flushing, hyperidrosis, lymph stasis, cell œdema, and finally, if it is sufficiently chronic, it may even result, as Unna has shown, in a keratinisation of the skin. Secondly, these cases have an inherent (probably inherited) weakness of their local peripheral *vaso-motor* centres, sometimes in one place, sometimes in another, so that their vaso-motor regulating apparatus is more easily and more readily upset than other people's. Such conditions as I have mentioned tend to occur more frequently in some people than in others, and often more in some families than in others. They also have a notable tendency to recurrence throughout life, sometimes, as in Amy P— and her brothers, to persistence throughout life. Then, in the third place, you have nearly always, at any rate in acquired conditions, some *toxic* blood condition which, owing partly to the sluggish circulation at the peripheral ends of the extremities, and partly to the vaso-motor defect I have just mentioned, remains longer, and therefore does more damage, at the dead end of the extremities. Sometimes you get one symptom, sometimes another of those I have been discussing. But in nearly all such cases one is able to trace all three of these factors in varying proportions.

Anomalous Œdema.

A curious localised and somewhat rare form of œdema is apt to affect the extremities, though it also occurs occasionally on other parts where loose cellular tissue is found. I have been accustomed to call it angio-neurotic œdema, and I am rarely without one case under treatment at the Skin Hospital, where there is a large clinique. It has been spoken of as anomalous œdema (which seems to be quite a good name), and by Weber and Bulloch as trophœdema (which seems to me not to be a good name). Some unnecessary confusion arises about the term "trophic," which should, in my opinion, be confined to those neuro-nutritive conditions such as anterior poliomyelitis, depending entirely upon cerebro-spinal nervous influence, whereas the condition under present consideration is undoubtedly vascular—trophic or nutritive, no doubt, in a sense, but nevertheless essentially vascular.

The anomalous œdema of which I speak consists of a localised patch of œdema, of rounded outline and sloping edges, occupying one side, or extending almost round the fingers or the limb, chiefly at the ends of the extremities, hands, feet, forearms, legs. The colour of the skin is generally white, but sometimes it is crimson or purple. It pits on firm, steady pressure, but not as readily as the anasarca of cardiac disease. There is no pain, only impeded movement. There are no evidences of thrombosis or renal disease. Each patch starts somewhat abruptly in the course of a few hours or a few days, and lasts an indefinite time of days (usually) or weeks (occasionally), varying in degree. Such patches have a tendency to recur at intervals throughout the first half of life, very often in the same position. In some cases both sides are involved symmetrically, but in others only one side. The condition may be associated with a tendency to recurrent urticaria, blueness of the ears and nose.

An interesting group of such cases of anomalous œdema was exhibited at the Clinical Section of the Royal Society of Medicine, December 12th, 1908, and these are summarised below. From the cases shown and the remarks made at that meeting, it would appear, as regards ætiology—(1) that more females are affected, (2) that it is very often congenital or starts early in life, (3) that there seems to be a distinctly hereditary tendency for the transmission of the disease through many generations, and that it is transmitted largely, if not entirely, through the female line. Dr. William Bulloch made exhaustive inquiries into the family histories of some ten cases of what he called "trophœdema," which supported the preceding statement. It would be interesting to know if the disease is associated with blue hands, cold hands, or white hands in the same family.

After a study of the cases that were shown on this occasion, I have been unable to detect any essential difference between these cases and those published under the names of Quencke's disease,

angio-neurotic œdema, inflammatory œdema, circumscribed œdema, urticaria œdematosa, œdème aigu, œdème bleu (Charcot), and œdème blanc. I have seen quite a number of cases described under these titles at my skin clinique, and the essential features of all of them are alike. Some cases are more acute than others, some cases have a greater tendency to affect the extremities, and others a greater tendency to affect the cellular tissues of the face and scrotum. In the 71 cases collected by Collins,* the first attack was noticed on the face in 29 instances, on the extremities in 22.

The cases exhibited were as follows :

(1) Congenital œdema of a family type (exhibited by Dr. G. A. Sutherland). A female child, æt. 18 months, had been the subject of pale œdema of the dorsa of both feet since birth. When the feet were cold they became blue. The hands were affected with similar swelling and occasionally cyanosis, and the mouth and ears became a little blue from time to time. Epistaxis had also occurred. Purpuric spots and large urticarial swellings in other places had been noticed. A sister, a child, æt. 6 years, was subject to similar swellings of the left leg since birth. There was no evidence of either cardiac or renal disease in either child.

(2) *Recurrent swelling of the left hand* (exhibited by Dr. E. I. Spriggs), in a female patient, æt. 18 years. In November, 1907, the backs of the fingers on the left hand swelled up. The swelling disappeared in the course of the winter. In February, 1908, the fingers and hand again became œdematous, blue, cold and tense. Pitting only came on prolonged pressure. The patient was treated by sodium salicylate, spiritus etheris nitrosi, on which she began to improve, the hand being quite normal by April 3rd. The swelling returned again in June, was well again by June 11th. On November 13th, 1908, she came again, aften an interval of five months, with the same condition of œdema.

(3) *Anomalous œdema* of the extremities (Dr. J. Rose Bradford). A woman, æt. 24 years, in whom a pale, œdematous swelling behind the right popliteal space came when she stood, and went away when she lay down. Gradual onset five years ago. She had also had progressive œdema in the right leg below the knee for eight years, and progressive œdema in the left leg for two years. In this case it seemed to me as though there was some loss of local compensatory power of the vaso-motor apparatus for standing and lying down. The heart and urine were quite normal.

(4) A case of *localised œdema*, first of one extremity, and then of another, in a male patient, æt. 21 years, was exhibited by Dr. H. D. Rolleston, which he suggested was due to wandering thrombosis. The urine contained albumen.

(5) Dr. Herbert French exhibited a case which he described as *trophœdema* of the right lower extremity; and (6) Dr. Parkes Weber exhibited a case that he called trophœdema of the left lower extremity. There seemed, however, some doubt whether these cases were not really instances of the semi-solid œdema, such as one finds resulting from thrombosis or other causes in early life, or of long standing.

A good deal of light might perhaps be thrown on vaso-motor affections by a closer study of such cases as these. There is no doubt in my mind about their being vaso-motor in origin. We know that œdema must be caused by variation in the size and permeability of the vascular channels. We know that when there is constriction, blocking, or increased pressure in the veins or lymphatics we get œdema. Similarly, when we get dilatation of an artery supplying a certain area, we get increased transudation through the walls of the arterioles and capillaries in the area supplied by that artery. Hence the shape and size of a localised œdema, which is evidently due to a dilatation of one or more arteries or arterioles supplying that area; or perchance a partial blocking of the veins or lymphatics which empty that area. In short, these are localised angio-neuroses affecting either the arteries (dilatation), or the lymphatics or veins (constriction).

February 15th, 1909.

* 'Amer. Journ. of Med. Sci.,' vol. civ, 1892, p. 654.

We have received from Messrs. Baillière, Tindall & Cox, London, a copy of the 'Third Report of the Wellcome Research Laboratories at the Gordon Memorial College, Khartoum,' by Dr. Andrew Balfour, the Director. This volume is published with a supplement containing a review of recent advances in tropical medicine, by Dr. Balfour and Dr. R. G. Archibald, which is replete with interesting information.

A CLINICAL LECTURE

ON

THE IMPORTANCE OF THOROUGH AND RATIONAL EXAMINATION OF THE URINARY ORGANS.

By R. P. ROWLANDS, M.S., F.R.C.S.,
Assistant Surgeon to Guy's Hospital.

I WISH to draw your attention to an interesting and difficult case, which may serve to make you realise the value of some of the modern methods of examination of the urinary organs. In this case a systematic use of these methods enabled us to distinguish between urinary and other diseases, to localise the urinary disease in the right kidney, and to decide upon its exact nature. They also proved that the left kidney was healthy and of normal functional capacity.

As a direct result of the examinations, a useless and perhaps dangerous anterior exploration was avoided, and a nephrectomy was confidently undertaken without fear of suppression of urine.

In this particular case something was learnt from the history, symptoms, physical signs, chemical, microscopical and bacteriological examinations of the urine, but the most valuable information was derived from the segregator, the cystoscope, and the estimation of the total amount of urea secreted in a given time. Radiography served to exclude calculus, but in other respects it was misleading.

In the short time at our disposal, I think that a careful consideration of this one case may be more instructive than a hasty review of many others which I could mention.

My sincere thanks are due to Dr. Lauriston Shaw for allowing me to publish the case, and to Dr. Joint for his excellent report.

Report.—A married woman, æt. 43 years, was admitted under Dr. Lauriston Shaw into "Clinical" Ward, Guy's Hospital, on January 28th, 1908, for repeated attacks of abdominal pain.

History.—She was the mother of thirteen children. Except for an attack of jaundice twenty years ago she had been remarkably healthy until about eight years before, when she first suffered from an attack of acute abdominal pain on the right side just below the costal margin in front. The pain was severe, doubled her up, and was associated with vomiting; it never radiated into the groin or thigh. Since then similar attacks used to occur about every other day for a fortnight, and then the patient would remain quite free of colic for months at a time, although she always had a sense of fulness in the right side. The last attack was three months ago. Jaundice had never been noticed in any of the attacks during the last eight years. The bowels had always been regular and the motions normal.

As far as she knew the urine had always been natural in colour and quantity. She had lost a good deal of flesh during the last two years. The appetite had been bad for months.

History of present attack.—A fortnight ago the patient suddenly developed one of her usual violent attacks of pain in the right hypochondrium, which so completely prostrated her that she had to take to her bed.

Hot applications to the abdomen gave but little relief. The pain was accompanied by vomiting and much flatulence.

The bowels were normal. There have been twelve attacks of colic during the last fortnight; the last was on January 26th. Since then there had been a dull aching pain under the right costal margin, but no acute pain or sickness. Just before the attack commenced the patient said that her feet and legs were swollen, but the swelling vanished with rest in bed.

Condition on admission.—"Temperature 97·4–100° F.; pulse 84–100; respirations 20–28. The patient is a thin woman with a dry, wrinkled, yellow skin, but she is not jaundiced. She has a slight aching pain in the right hypochondrium. Here a slight fulness can be seen, and a swelling which moves very little with respiration can be felt. The swelling consists of two parts, a large elastic mesial one which extends towards the left as far as the middle line, downwards to the level of the umbilicus and upwards to the liver, to which it seems to be connected. Its outlines are indefinite, but it appears to extend deeply towards the spine; some of it is moderately resonant on percussion. Towards the right it merges into a more solid dull mass, whose dulness is continuous with that of the liver (which extends upwards to the sixth rib). The outer swelling is tender and appears to be adherent to the abdominal wall at one spot. It cannot be moved laterally. The tumour can be

felt bimanually through the loin, which is dull posteriorly and at the side.

"The spleen is not palpable. A loaded sigmoid colon can be felt. No other abnormality can be discovered in the abdomen or by rectal examination. The urine is normal except for a trace of albumen."

On February 3rd the abdomen was skiagraphed, and the radiographer's report is as follows:

"Nothing abnormal is seen in the abdomen; the left kidney is well seen being shown up by gas in the descending colon; it is normal. The right kidney is not visible, being probably obscured by soft fæces in the ascending colon."

February 6th: The patient had been having more attacks of pain, principally at night time. She had several times been sick with the pain. Since admission she had been rather constipated, and had required aperients every night. The lump appeared to be more definite.

The general condition improved a little, and the temperature remained normal for several days.

On the 11th slight irregular pyrexia again developed.

On the 13th the bowels were well cleared out, and the patient was again skiagraphed, the report being as follows: "The kidneys appear normal in size, shape, position, and mobility. There is a dark shadow continuous with the liver shadow and appearing to be an extension downwards of the right half of the liver. There is no definite lower border to this shadow. The diaphragm and the bases of the lungs appear normal. No gall-stones are seen. They are scarcely ever visible, being transradiant. No other abnormality found."

On February 15th a little pus was discovered in the urine.

On the 17th it was believed that the patient probably had malignant disease of the gall-bladder, associated with gall-stones and suppuration.

I was thereupon asked to see the patient, and in view of the attacks of colic, and the history of scanty and frequent micturition during the attacks (which the patient had denied on admission), and the presence of a little microscopic pus in the urine, I suggested that the trouble was renal and not biliary. I wished the urine to be measured for a few days, so that the total amounts of urine and urea could be calculated. I suggested that a catheter specimen should be examined, so that the possibility of the pus coming from the vagina could be eliminated.

February 18th: A catheter specimen of urine showed the presence of pus, but in smaller quantities than before.

February 21st: The average amount of urine passed per twenty-four hours during the last three days had been only 21 ounces, and the average amount of urea in the same time had been only 140 grains in the twenty-four hours.

Cystoscopy showed fasciculation of the bladder, with chronic cystitis, with dull mucous membrane, so that it was difficult to identify the right ureteral orifice with certainty; but urine issued from the left ureter. Upon segregation, no urine escaped from the right tube, in spite of bimanual pressure upon the loin. There was no pus in the urine that came from the left kidney.

On the 22nd: A large amount of pus appeared in the urine to-day, probably due to the shaking and the sitting-up position assumed for segregation There was no pain or discomfort in the bladder.

February 24th: *Operation.*—The right kidney was exposed through the usual oblique posterior incision, which had to be made eight inches long. A very large lobulated kidney with a greatly distended pelvis was found. At one spot in the flank it was firmly adherent to the abdominal wall extraperitoneally, and while completing the separation at this part the contents of the kidney began to escape. The kidney was immediately drawn well out of the wound and packed round with gauze to prevent the pus escaping into the loin. The renal vessels were ligatured and divided, and the ureter was then liberated for several inches until a healthy part appeared. It was then clamped, ligatured, and severed.

The wound was closed except at the posterior extremity, where a drainage-tube containing a wick of gauze was inserted. The operation lasted nearly half an hour. To combat shock the foot of the bed was raised and the patient's legs were firmly bandaged over cotton-wool. Ernutin ℥v was injected subcutaneously every four hours, and one pint of normal saline solution was given *per rectum* every three hours. In spite of these precautions there was considerable shock after the operation, and the patient vomited at intervals for thirty hours. During this time she retained about eight pints of saline solution given *per rectum*.

After thirty-six hours the foot of the bed was lowered as the pulse was steadily improving. No urine was passed for twenty-four hours; during the next day she passed only four ounces. Afterwards the amount increased rapidly; on the third day she passed sixty-eight ounces, and on the fourth ninety-two ounces. The pus gradually and completely disappeared from the urine.

The drainage-tube was left out after a week, and the wound rapidly healed. The appetite improved, so that ten days after the operation the patient stated that she had not been so hungry for years.

On April 2nd the patient left the hospital feeling and looking very well.

Pathology.—No organisms could be cultivated from the pus contained in the kidney. The kidney and its pelvis were enormously dilated, and the cortex was thin and grey. At one spot, where it had been adherent to the parietes, it had ulcerated through.

The ureter and the lower part of the pelvis were curiously kinked, like a concertina, so that the lumen was obstructed even when the pelvis was not distended. The kinked condition was maintained by a good many peri-ureteral inflammatory adhesions. There was no sign of stone.

Sections of the kidney showed chronic inflammation with but little renal tissue. There was no evidence of tuberculous disease. The lower part of the ureter was healthy.

It is certain that the dilation of the pelvis was due to kinking at the commencement of the ureter. The cause of the kinking is more obscure; it may have been originally due to mobility of the kidney. No abnormal vein was seen to compress the ureter. It is possible that the valvular arrangement may have been congenital, but exaggerated by peri-ureteritis secondary to distension of the pelvis and repeated attacks of septic inflammation.

I want you to let this case remind you that rational treatment must be based upon accurate diagnosis, and that accurate diagnosis can only be made by carefully weighing all the available evidence, without paying too much attention to any part of it, however fascinating that may be. You should spare no efforts in your endeavour to collect reliable facts, and to do this you must become proficient in the modern methods of investigating urinary disease. This requires a good deal of practice.

For some time this patient was supposed to be suffering from malignant disease of the gall-bladder, with secondary suppuration. The history of jaundice and of repeated attacks of colic in the right hypochondrium seemed to support this view. It was thought that malignant disease had developed as a result of the chronic irritation of stones lodged in the gall-bladder or cystic duct. The hardness and adhesion of a part of the swelling were considered ominous. The tenderness and intermittent pyrexia pointed to secondary suppuration. Upon careful re-examination, however, it was noticed that the dull part of the tumour was unusually far out for the gall-bladder, and that the mesial part was unlike the gall-bladder, because it was not completely and constantly dull on percussion.

Carcinoma of the hepatic flexure of the colon with secondary localised peritonitis was considered, but excluded on account of the absence of intestinal symptoms, except for some flatulence and constipation, which could easily be explained in other ways.

The swelling was placed too far to the right and too high for a pancreatic cyst, and the absence of jaundice and excessive wasting were also against other neoplasms of the pancreas. Disease of the right kidney was at last suspected, because of—(1) The general resemblance of the shape of the tumour to that of the lower end of a greatly distended kidney and pelvis; (2) the variable area of resonance in front of some of the mesial part of the swelling, which was thought to be the dilated pelvis partly behind the colon; (3) the tumour could be felt bimanually through the loin, but this was not easy on account of rigidity.

The fixation of the swelling and its apparently even continuity with the liver in front, the X-ray reports, and the history seemed to be against a diagnosis of kidney disease.

The history first obtained had been very misleading, but on careful cross examination it was ascertained that frequent and scanty micturition used to accompany the attacks of colic, and that a large quantity of urine had been voided after some of these attacks.

The right ureter must have been completely blocked when the woman was admitted, for no pus was found in the routine examination of the urine. Later on, however, when a microscopic examination was made, pus cells were discovered.

To exclude a vaginal source of the pus a catheter specimen was then examined, and microscopic pus was still found. This fact was of great importance, for it encouraged us to estimate the average total amount of urine and urea passed during twenty-four hours. The average for three days was taken, for nothing less than this is of much value.

The average amount of urine was only twenty-one ounces instead of about fifty. The urea was only 140 grains instead of over 300.

Thus it became clear that one or both kidneys must be diseased.

The X-ray reports were, in some respects, misleading, although they were valuable in excluding stone as the cause of the trouble. Remember that it is always necessary to screen the whole of the urinary apparatus in order to get reliable information. In hydronephrosis and pyonephrosis the X-ray appearances may be puzzling, as in this case, because of the great increase of size of the comparative opacity, which is less dark than the normal kidney. When the opacity is examined as a whole its shape may be seen to correspond with that of the lower parts of the dilated kidney and its pelvis.

Do not forget the need of thoroughly emptying the colon before sending a patient for X-ray examination, and bear in mind the value of repeating these examinations, which should nearly always precede cystoscopy and segregation.

Cystoscopic examination alone was not conclusive in this case, because of the chronic cystitis and fasciculation. This made it difficult to recognise the right ureter with absolute certainty, especially when nothing escaped from it. The subcutaneous injection of a sterilised solution of sodium sulphindigotate enables us to see coloured fluid issuing from the ureters. It is far easier to watch this than the ordinary urine.

Segregation, by showing that no urine escaped from the right ureter, proved that the swelling was a dilated right kidney. The nature of the obstruction of the right ureter was not so clear. Stone was very unlikely because two X-ray examinations had failed to show one. It was just possible, however, that a small uratic calculus in the ureter might have been invisible, but it is extremely rare for our excellent radiographers to overlook a calculus. The repeated attacks of colic were against tuberculous disease. Bacteriological examinations of the urine would have afforded additional evidence. Calmette's reaction was negative. There was no time for inoculation experiments. The evidence seemed to point to some form of valvular obstruction or stricture of the ureter near the kidney. It was considered probable that kinking had resulted from a movable kidney, with secondary dilation of the pelvis and increase of kinking. The fixation of the kidney and the tender area were thought to be due to spreading of inflammation into the subperitoneal tissues.

The absence of pus and casts in the urine derived from the left kidney and the knowledge that the right ureter was completely obstructed, were of great value, proving, as they did, that the left kidney was healthy and already doing all the work, so that the right kidney could be excised without hesitation.

The slight amount of pus discovered in the urine was easily explained by the persistence of chronic cystitis, the result of earlier infection from the right kidney.

A few remarks may be made about the operation. I think it very important to take the kidney out without opening it, thus avoiding contamination of the wound. This can be done by carefully separating the kidney before attempting to deliver it, and by making the wound large enough. Before enlarging the wound forwards, care must be taken to displace the peritoneum forwards out of harm's way. It is important to remove all the diseased part of the ureter, clamping and ligaturing before severing it.

Elastic pressure upon the extremities is a simple and useful measure against shock. Ernutin and saline solution given subcutaneously or *per rectum* are the best means of combating shock. Drainage of the loin for two or three days is wise after all nephrectomies.

So far, out of my twelve nephrectomies all the patients are alive and well except one who died from lobar pneumonia.

February 15th, 1909.

MESSRS. CASSELL & Co. have sent us the fourth edition of 'A Manuel of Modern Treatment or Clinical Therapeutics,' by Dr. Burney Yeo, Dr. Raymond Crawfurd, and Dr. E. Farquhar Buzzard The work is in two volumes, and has been brought well up to date.

THE SURGICAL TREATMENT OF TUBERCULOSIS OF THE EPIDIDYMIS AND TESTICLE.

By CHARLES GREENE CUMSTON, M.D.,
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DURING the past few years the treatment of tuberculosis of the seminal gland and its adnexa has given rise to much discussion. In 1899 the Surgical Society of Paris considered the question of surgical treatment very fully, while in 1907 an important paper on "Epididymectomy" was read and discussed before the New England Urological Society. In going over the reports of these meetings there is one point upon which accord seems unanimous, namely, that castration should never be resorted to until all other treatment has proved useless. Bilateral castration, as is well known, sometimes gives rise to very serious mental disturbances. These are not due simply to the mental effect on the patient from his knowledge that he has lost both seminal glands, but the loss of the genital functions and sexual impotence are factors of far greater importance. Then, again, the internal secretion of the glands must occupy a very prominent place, for it is well known that the testicle possesses a function quite independent of the spermatic secretion, a function controlling, to a certain extent, the organic equilibrium.

The exact nature of this function is as yet rather obscure, but, nevertheless, it seems undeniable. It is commonly admitted that every glandular structure having an external secretion, when deprived of its excretory ducts becomes atrophied, while the testicle, on the contrary, when deprived of its excretory duct, retains its normal structure and size, and shows no evidence of an atrophic process. A loss of virility has been noted following diseases which suppress the functions of the testicle, such as the orchitis of mumps or syphilis. Contrary to gonorrhœa, tuberculosis is particularly prone to attack the epididymis and never gives rise to impotence.

Another and, perhaps, more important fact, is that patients who have been deprived of the excretory ducts of both testicles have, for all that, retained their genital faculties. They are sterile but retain their virility, whether the solution of continuity of the excretory ducts is pathological, as occurs in tuberculosis and gonorrhœa, or of a traumatic or operative type as occurs, for example, in bilateral epididymectomy. It is rather difficult to establish a demarcation between the disturbances produced in the organism from the removal of, and those simply arising from the moral effect produced by the loss of, the testicles, but in practice this is really of no particular import. But there is one point upon which I would insist, namely, that if one testicle or even a small portion of one gland is left, the internal secretory functions will continue. It is for this reason that many surgeons have advised early castration in cases of unilateral infection, and several years ago I was inclined to coincide in this view, as will be seen by my paper published in the 'American Journal of Medical Science,' June, 1904. Although I still believe much of what I said therein, I nevertheless must confess that since I have adopted epididymectomy the results obtained have been so encouraging that I feel this is the proper surgical procedure *when the seminal gland itself is not involved in the process—a fact which can only be ascertained by first splitting open the testicle.* If, then, the latter is found free from disease it is sutured, and the epididymectomy proceeded with. I must lay particular stress upon this exploratory incision of the testicle, which I referred to at the New England Urological Society a few months ago, when demonstrating some specimens of tubercular epididymitides removed surgically.

Berger has said that the question of castration in tuberculosis of the testicle may arise when only one gland is involved, usually in the early part of the process, when the lesion in this organ can be proven to be the only tubercular manifestation in the patient, and he is of the opinion that under these circumstances castration represents a radical measure by which an isolated focus of tuberculosis may be removed, that in other words it is curative. Consequently the partisans of early castration resort to this operation even when the epididymis is only apparently involved, believing that the testicle is also often invaded even when it appears intact, but, as I have pointed out, if exploratory incision of the organ is made the question is at once settled. The partisans of castration hope, by radically removing the tuberculous focus, they will prevent extension of the disease to other

viscera, and particularly the remaining testicle. In defence of their attitude they say that after castration, in cases where lesions of the prostate and seminal vesicles exist, the latter undergo regression after castration, while their last argument is that it is useless to preserve a testicle which has lost its power of spermatogenesis, this function as well as that of the internal secretion being amply fulfilled by the remaining testicle. which it is hoped will remain healthy. In my paper on "Castration for Tuberculosis," already referred to, I quoted a number of surgeons who had never seen the remaining testicle become involved after an early castration of its diseased fellow, while others say that, should the second one become involved, a conservative treatment can always be resorted to.

Let us now examine the arguments put forward by those believing in conservative measures. In the first place they state that an early unilateral castration is justified if it could surely arrest the progress of the tuberculous lesions, and if it could positively assure immunity to the remaining organ. But this is far from being the case, and no matter how extensive and complete be the effect of the operation, one can never be sure that all the tuberculous lesions have been done away with, which are frequently secondary to lesions of the same nature in the lymph-nodes or viscera, and are unrecognised at the time of the operation. It is more than probable, I believe, that many patients have lesions of the same nature in other organs, and to my way of thinking, if this is the case, this very fact takes away all curative and prophylactic value of castration.

It is known that, generally speaking, it is by way of the prostate and seminal vesicles that tuberculosis of the genital glands makes its invasion, and no matter how fine the results obtained by Berger and others by early unilateral castration, it is to be supposed that they have had to deal with a particularly fortunate series of cases, because numerous are the instances in which early castration has not prevented the development of the disease in the opposite testicle. It should also be pointed out that the warmest partisans of early radical interference advise a conservative treatment if the remaining testicle becomes in its turn involved, and it may be questioned why one should wait for this to occur to put in practice this treatment with a greater chance of success, since only one testicle remains. This method is not only a dangerous one, but it is likewise useless, for the simple reason that the best results obtained by it are no better than those derived from less radical measures. I believe I can safely say that recurrence after epididymectomy is no more frequent than when castration is done, while its influence on the patient's general condition is quite as good and its action on a diseased prostate or seminal vesicle quite as marked.

Operations on the epididymis alone have the same action on the regression of prostatic and vesicular lesions as has castration, as has been said, and no lesser authority than Debbet has said that he has never seen an abscess of the prostate or seminal vesicle occur after the conservative operation has been done. The rapidity of some post-operative improvements has even led him to question whether or not the symptoms considered as characteristic of tuberculosis of the prostate and seminal vesicles were not in some cases simply due to congestion of these organs. But in those cases where the tuberculous lesions of the prostate and vesicles were undeniable, he is led to suppose that the virulent products of the tubercles in the epididymis constantly spread over the upper spermatic tract, and that doing away with these bacterial discharges by removal of the epididymis result in their cure.

However this may be, such improvement certainly does occur after epididymectomy, and for this reason cannot justify castration. This first point appears to be settled, but there is another upon which opinions seem to be in accord, and still the conclusions are absolutely in opposition; I refer to acute tuberculosis of the testicle. In this form, which at the commencement is usually distinctly localised, early castration is advised in order to prevent rapid extension and the dangers of generalisation which are present, all the more so because the testicle usually becomes eliminated by purulent disorganisation when surgical interference is not resorted to. Now if, generally speaking, castration is to be condemned in cases where the lesions of chronic tuberculosis are beginning, and if, on the other hand, radical operation is a necessary evil in acute tuberculosis and in certain hopeless cases, there are cases of medium intensity in which the testicle itself is markedly involved, and for which

many surgeons advise castration. I shall refer to this question later on.

Tuberculosis of the epididymis, with or without involvement of the seminal gland, is a distinct type of local tuberculosis, and if in a number of cases it occurs in subjects who are otherwise tuberculous, there are also numerous instances where the patient is attacked in a state of perfect general health, and may probably never present any other tuberculous lesion, at least one that is clinically appreciable. The researches carried out by Reclus, which are both pathological and clinical, have led to the following conclusions: In 50 per cent. of the cases the lungs were not involved clinically. The results of the autopsies were somewhat different, because in only 33 per cent. the lungs were not involved, and the same conclusions are to be found in Villard's statistics, which show that the testicle alone was involved in 30 per cent. of patients autopsied. From this it will be seen that the dictum that tuberculosis of the testicle will sooner or later extend to the other organs is not by any means absolute. In point of fact, in many cases genital tuberculosis develops and runs through its clinical course without involving any other organ, while it frequently presents a tendency to spontaneous cure, this being the result of the defensive reaction of the tissues. From this it may be concluded that surgical treatment has its indications, and will very frequently give very excellent results.

Much discussion has been given to the question as to how the bacillus reaches the testicle, and leaving aside those cases in children where the infection is directly transmitted from the peritoneum to the vaginalis by way of a patent vagino-peritoneal canal, and on the other hand rejecting the theory of ascending infection by way of the urethra, it may be admitted that in the adult tuberculous infection of the testicle is either transmitted by a descending epithelial infection or by way of the blood. It seems to be an acquired fact that the testicle may be invaded by the extension of tuberculous lesions in the kidney or bladder, in which case the bacillus follows the course of the urine and then descends that of the sperm. In tuberculous cystitis it is still an open question whether or not latent prostatic lesions are the first to appear, but in the majority of cases it is by way of the blood that the infection occurs, whether it attains directly the testicle itself, or, as is more generally admitted, it in the first place invades the prostate and the testicle secondarily by the vas deferens. Thus is explained why the epididymis is always involved before the testicle, and the importance of this pathologic notion from the therapeutic standpoint is easily conceived, because an early interference will reveal lesions localised to the epididymis only, and its removal may be resorted to without touching the testicle.

The tuberculous lesions arising in the testicle of the adult commence always in the epididymis, and Reclus' statistics are conclusive in this respect, because in numerous cases he only once found the lesions limited to the testicle. Now, if the epididymis is the first involved, it nevertheless must not be forgotten that the infection does not remain localised to this structure, and very often the testicle is involved at the same time, as will be seen by the following figures given by Reclus; out of sixty-eight autopsies both the epididymis and testicle were involved in forty-eight subjects, the epididymis alone in twenty. Thus in about two thirds the testicle and epididymis were simultaneously involved.

If now we consider the clinical aspect the proportion will be found reversed, because in only about 33 per cent. of patients did the testicle appear involved. This is easily explained from the fact that in the epididymis the tuberculous infection presents itself from the very commencement in the form of caseous masses, which are easily palpated, while in the testicle only granulations arise which do not change the anatomical condition of the organ when palpated. If caseous masses do exist they are usually found in the body of Highmore, adherent to the epididymis, from which it is impossible to differentiate them. From the practical standpoint the impossibility to make this distinction is of no value.

From the clinical point of view the disease may appear in two very different aspects; in the first, which corresponds to the period of development of the tubercles, a swelling, formed by one or several hard foci, is felt, while more or less pain is elicited by pressure. In other cases the entire epididymis is involved and may reach two or three times its normal size, while the seminal gland itself is normal to the feel and not enlarged.

The functional disturbances, usually absent, are those symptoms arising in the bladder and prostate, these being frequent desire to urinate, a mild

degree of vesical tenesmus, slight hæmaturia, a sero-purulent urethral secretion, etc. These symptoms would seem to indicate an early participation of the prostate in the infection, and in point of fact, if rectal examination is made at this time one will usually encounter tuberculous nodules in the prostate and seminal vesicles. But during all this time the tissues subjacent to the testicle present no changes, the skin is normal in colour, movable over the testicle, and only a slight hydrocele occasionally indicates a reaction in the vaginalis.

On the contrary, when the lesions are to end in suppuration, pain will be complained of, and characteristic local symptoms will be observed. At some point in the scrotum, usually on its posterior aspect, that is to say, where it is in direct relationship to the epididymis, the envelopes of the testicle will be found to have lost their normal mobility on account of the adhesions with the gland; the skin becomes reddened, and a point of fluctuation will be found, this finally opening and giving exit to caseous material. A fistula then results, giving exit to a certain amount of secretion for an indefinite period, sometimes momentarily closing up, only to open later. In some fortunate cases it will end by closing for good, leaving behind a cicatrix adhering or not to the testicle by a fibrous cord. But one thing to be particularly remembered is that very frequently, *in spite of several fistulæ and the persistency of suppuration, the lesions may, nevertheless, remain localised to the epididymis.* A number of such cases, including one of my own, prove this, where, after more than a year of suppuration, the epididymis was found alone involved and the testicle itself healthy. The patient, æt. 33 years, was referred to me for two fistulæ on the right side of the scrotum. The testicle on this side had been enlarged for four months; then an abscess formed, which opened, after which the fistulæ remained. Several months later another abscess formed, this being incised by his physician. The fistulæ continued to secrete a certain amount of tuberculous matter. When the patient came under observation the right side of the scrotum was red, and posteriorly two fistulous openings were found. By palpation the right testicle was with difficulty made out from the epididymis; the latter was very much enlarged and painful on pressure. The vas deferens was slightly enlarged and hard to the feel. The prostate did not appear enlarged, its surface was regular, and no pain could be elicited on pressure. In other respects the patient's health was perfect, and several days later epididymectomy was done with free removal of the vas, and at the operation the testicle itself was proven to be absolutely normal by an exploratory incision. The patient made an excellent recovery, and was perfectly free from any trouble nine months after the operation.

In tuberculosis of the epididymis one can be extremely radical in all operative interference, because the organ simply represents a single tube rolled up on itself, and as far as excretion of the sperm is concerned it has lost this function from the fact that its lumen has become permanently obliterated by the tuberculous process. Therefore the removal of the entire organ will in no way change the condition of affairs from what it was before the interference. This statement applies equally well to bilateral epididymectomy. This means that although I now reject early castration for the reasons already given, I do not believe that it is necessary to hold to merely a general treatment as some writers advise when the lesions have not gone on to suppuration. In many instances this treatment will cause a regression of the lesions, but one should not lose sight of the numerous cases where the process has a tendency to extend when an insignificant operation might have been quite sufficient to have completely controlled the situation.

Many methods for destroying the epididymis have been advocated in the past, but to my way of thinking clean and decent surgery in case of the epididymis, as with salpingitis, resides in complete enucleation. And in point of fact a tuberculous epididymis is in reality a miniature pus-tube. Therefore I will limit my remarks here to the technique of epididymectomy as I perform it. The incision should be made on the anterior aspect of the scrotum to the extent of two or three inches, and should include all the structures of the scrotum, comprising the vaginalis, so that the testicle is at once freely exposed. Next, the testicle itself is split open and thoroughly examined, and if found healthy it is sutured with fine catgut, *not* chromicised, and the removal of the epididymis is then proceeded with. The separation of the epididymis should be begun at its tail, and all that is necessary is to incise the vaginalis both inwardly

and outwardly in order to decorticate the organ from the testicle. A little further up the fold is cut which unites the middle portion of this organ to the posterior superior border of the testicle. The vas deferens is cut, but care must be taken in doing this not to injure the internal branch of the spermatic artery which reaches the testicle at this point. Consequently, one should nick the vaginalis at the internal aspect of the head of the epididymis, and then, the vas deferens having been cut, the epididymis is drawn upwards and simply peeled off from the vascular mass. When this is done the epididymis is simply retained by a few small vessels, the vas deferens and cellular tissue, and, a ligature having been applied to this mass, a stroke of the scissors completely frees the organ. Any bleeding should be controlled, and after this a small cigarette drain in contact with the testicle is inserted and the scrotal incision closed. The drain may be removed at the end of two days.

This technique is proper when the epididymis alone is involved, and even in certain cases where the lesions involve only a portion of the epididymis partial resection of the organ may be resorted to; but I do not advise this, believing, for the reasons given above, that total epididymectomy is the only proper procedure. Certain surgeons have endeavoured to form an anastomosis between the vas deferens and the testicle after epididymectomy, and for those desirous for more ample detail on this subject I would refer them to my paper on "Castration for Tuberculosis of the Testicle," to which I have already referred.

Now, if the body of Highmore looks suspicious after the epididymis has been removed it should also be excised. If the vas deferens shows evidence of disease it is absolutely necessary to resect it as far up as possible, and as it may be followed down into the peritoneal cavity for a considerable distance by means of an operation that I devised a number of years ago for the radical removal of malignant disease of the testicle, and as this technique seems to me the best that has been so far offered, I would refer those desirous of knowing the steps of this interference to Casper's 'Text Book' (American edition), or Binnie's 'Manual of Surgery,' in which it is described, or to my original paper, which appeared in 'American Medicine,' April 18th, 1903.

In those cases where the epididymis is adherent to the posterior aspect of the scrotum, or if an abscess has opened in this region, the organ should be reached at this point instead of by an anterior incision. If necessary the diseased skin and structures may be resected, the fistulæ likewise excised freely, in order to avoid dissemination of the infection during epididymectomy.

The conservative operation of epididymectomy for tuberculosis has given so far a really large number of both successful and permanent results, but I would now consider the circumstances which would force one to resort to the more radical operation of castration. In the first place, an indication for castration would be a failure to cure the process by epididymectomy. Curettement and cauterisation in cases of recurrence may be resorted to, but if these fail to control the process, if fistulæ persist, the testicle must be sacrificed in order to do away with the prolonged suppuration. It is useless to endeavour to preserve the seminal gland in these cases, and the earlier the radical interference is undertaken the better it will be for the patient's general health. I also believe that castration is absolutely indicated in patients who present advanced pulmonary lesions, because the local lesions in the genital apparatus will have no tendency to disappear after a conservative operation, and these subjects cannot stand the debilitating effects of a prolonged suppurating process. However, I would point out that if the patient is well-to-do, and can live in Colorado or other resort, an unexpected cure may sometimes be obtained without operation, but recovery from a genital tuberculous infection can never be surely counted on from climacteric treatment, and therefore in most instances operation is advisable.

I would like to say here a few words relative to the acute type of tuberculosis of the epididymis and testicle. Its characteristic is *the intensity of the symptoms presented at the commencement of the process*. In a very few days the testicle becomes the seat of extreme pain, while at the same time a tumefaction appears. Palpation shows that this tumefaction has a perfectly smooth surface, and includes the epididymis particularly, the testicle can be made out separately, at least at the commencement. This, however, is not absolutely true of all cases, because both organs may be equally involved, forming a single mass, and careful palpation will not allow one to distinguish between the

epididymis and the seminal gland. The tumefaction may become very considerable, while a certain amount of hydrocele, redness and œdema of the scrotum, indicate the participation of the vaginalis and skin in the process. There is a local elevation of the temperature, likewise that of the general temperature.

But what is to be particularly noted is that in *the acute form the prostate and seminal vesicles remain uninvolved in the beginning of the process.* After a few days of the acute symptoms a period of remission occurs. The general symptoms disappear, and the affection then in no way can be differentiated from the ordinary type other than by the rapidity with which the tuberculous lesions made their appearance and progress. Within a few days or a few weeks suppuration will invariably occur, the pus burrowing outwards and giving rise to interminable fistulæ, which give exit to the broken-down testicular tissues. The general condition of the patient is quickly affected by the suppuration, and at about this time prostatic and vesicular lesions denote that the infection, which was in the first place localised, has extended beyond its primary limits.

In the treatment of these acute cases a conservative operation is absolutely out of the question, and castration is fully indicated at the earliest possible moment. I have referred to my technique of castration, which is a very radical one, and in closing this paper I will quote from my article already mentioned on "Castration for Tuberculosis of the Testicle":

"Von Bungner has pointed out a fact which is well known, that the spermatic cord is often diseased without presenting much evident infection at the time of operation. From this arises the danger that the diseased parts which are left behind in the cord will act as disseminating foci for further tuberculous infection, and, in order to avoid this danger, he recommended a new method, which he termed 'castration with evulsion of the spermatic cord.' As in castration the spermatic cord is separated from the vessels and its surrounding coverings, the vessels are tied off below the external inguinal ring, and then the isolated vas deferens is slowly but steadily pulled upon until it breaks. The piece of vas is removed along with the diseased testicle and epididymis. In his operations von Bungner was able to extract about four fifths of the spermatic cord by his method of evulsion, and, according to his statistics, he obtained a cure in 50 per cent. of his cases.

"It is quite evident that removal of the vas deferens should be undertaken as far as possible in every case of castration, especially when it is evidently diseased, but the method of evulsion is certainly not devoid of danger. Lauenstein and Helferich pointed out the danger of hæmorrhage occurring in the pelvic tissue from the traction exercised on the vessels accompanying the spermatic cord. The first-mentioned authority performed castration with evulsion of the vas in thirteen cases, and in several succeeded in removing quite large portions of the cord, with much benefit to the patient, but in two instances such severe hæmorrhage arose that it was extremely difficult to control it. Since this time Lauenstein only removes as much of the vas deferens as can be pulled down without exercising traction upon it.

"A point which should not be forgotten is that in the method of evulsion the peritoneum can be easily torn, and with the raw surface of the cord the peritoneal cavity might easily become infected. Consequently the method of evulsion is certainly not devoid of danger, and it also should be remembered that in all probability the diseased spermatic cord will rupture at a point where it is infected by the tuberculous process on account of lesser resistance. If this be true—and there is every apparent reason that it should be so—it would naturally scatter tuberculous material, which would be the starting-point for a new dissemination of the disease. For these reasons Schede employs a technique similar to that described by Lauenstein, dissecting the spermatic cord up as high as possible and then ligating it at a point where it appears to be healthy, and, for this purpose, it may be necessary to dissect the peritoneum off the cord to a certain extent. It is evident that in this operation, as in others, the spermatic cord is not always removed beyond the point of disease, and consequently the affection still remains in the intra-abdominal portion of the vas which is left behind. For this reason it becomes evident that the removal of the vas in its intra-abdominal portion must be accomplished under the direction of the eye, and this can be done if my technique for the radical cure of malignant disease of the testicle be correctly employed.

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ON THE SIGNIFICANCE OF FEVER IN PATIENTS FROM THE TROPICS.*

By SIR PATRICK MANSON, K.C.M.G., M.D., F.R.C.P., F.R.S.,

Physician Seamen's Hospital Society.

MR. PRESIDENT, LADIES AND GENTLEMEN,—When your secretary, Mr. Elliott, asked me, I believe at the instigation of Mr. Godlee—a somewhat unfriendly act—to read a paper before this Society, I was obliged to decline, on the ground of want of time, and possibly also want of adequate material for so formidable an undertaking. But I said I would be very glad to give a talk to the Society. And in casting about for a subject I thought I could not do better than speak about something that I know, if you properly apprehend my meaning, would be of enormous practical value to you in your future work, a subject which I ought to be acquainted with, at all events I have had abundant opportunity of becoming acquainted with it, a subject connected with tropical disease, which, as you know, is yearly becoming of more importance, not only from the standpoint of general medicine and pathology, but also from that of practice. For year by year the British Empire in the tropics is expanding, and every year more men and women work in the tropics, and more invalid men and women return from them, so that I fancy hardly a practitioner in the kingdom fails once or twice, or oftener, every year to come across cases of tropical disease, or at all events, of patients who have returned from the tropics suffering from some disease.

There are two principal diseases, from the standpoint of the patient, that are brought under the notice of the practitioner. One is intestinal flux, and the other is fever. Patients from the tropics

* Address delivered at the University College Medical Society.

come to you complaining of one or other of these conditions.

This evening I propose to speak about the significance of fever in patients coming from the tropics. The question of intestinal flux I shall leave for someone else to talk about.

You will find that, as a rule, your diagnosis has been already made for you—a patient coming from the tropics with fever tells you that he is suffering from malarial fever. He does not want you to diagnose his disease, he only wants you to treat it. Now, if you take his diagnosis for granted as being correct, take my word for it, in nine cases out of ten you will make a very grievous mistake. For this reason: malarial fever, of course, is exceedingly common in the tropics; a man who has been in the tropics expects to get malaria, in fact, he is rather disappointed if he does not get it, and if he does get any fever, because malaria is extremely prevalent in the tropics, his idea is that the fever which he has is necessarily malarial. That is a great mistake. There is one axiom I have learnt, and it is this, never trust the patient's diagnosis. And another axiom which I have learnt is, never trust a diagnosis of malarial fever, because in nine cases out of ten it is wrong. The difficulty of interpreting the significance of fever in tropical disease is in great measure imaginary, arising from our own preconceived ideas as to diagnosis and the preconceived ideas of patients as to diagnosis. In olden times, as regards other subjects than malaria and fever, our notions of an Indian, or a European from India, were exceedingly erroneous; we thought that every man coming from India had a big liver, a big appetite, a big spleen, and a bad temper, and was jaundiced, bilious, and a gourmand. We know now that that description is erroneous; we have overcome that notion about the Anglo-Indian.

My rule in interpreting the significance of fever in a patient from the tropics, my first rule, which I commend to you, is to ignore the fact that the patient has been in the tropics. Do not look upon him as a man from the tropics while you are trying to arrive at a diagnosis, but as an ordinary patient, and before you conclude that he has a tropical disease eliminate every possibility of an ordinary vulgar European disease. And take care to do that systematically, for, if you do not, sooner or later you are sure to trip. Think of tuberculosis, think of syphilis, think of malignant disease, think of local diseases; begin at the crown of his head, and end at the sole of his foot, questioning every organ as to whether it is in a normal condition or not. And do not forget his frontal sinuses; I have seen more than one case of frontal sinus disease regarded as malarial disease because the condition was accompanied with an intermittent fever with a daily rise of temperature, possibly with a rigor ending with a sweat. Not many years ago I was asked to see a patient at the west end of London who was said to be suffering from Roman fever. Popular ideas about Roman fever are very strange. Everyone seems to assume that if a man goes to Rome he gets fever, and that everyone who gets Roman fever has malarial fever. As a matter of fact there is no malaria in Rome, it is outside Rome that people get malaria. The people who get fever in Rome itself get typhoid. But this particular patient had been in Rome and had come back with an intermittent fever. He had fever every afternoon, a bit of a rigor followed by a heightened temperature ending in sweating. And this went on week after week for months. As a result the patient was emaciated and presented a hectic appearance. Several physicians had seen the patient; one thought it was tuberculosis, another thought it was typhoid, while another considered it likely that it was Mediterranean fever. Finally I was sent for, because it was assumed by someone to be a case of malarial fever, and the patient had been crammed with quinine on that assumption, and I was asked whether the doses of quinine should be increased. I examined the patient carefully, but I failed to find any evidence of malarial fever. He had fever, and he had come from Rome, but there was no pathognomonic sign of malarial fever present (these signs I shall describe presently), and, as I recommend you to do when in doubt, I said I did not know what it was. I said to the patient's friends, "I do not know what is wrong with him, only one thing I am sure of, and that is that he has not malarial fever." Three or four weeks afterwards I met a friend who interests himself in noses, ears and throats, and in the course of conversation he said, "You saw Mr. So-and-So," naming the patient. I said "Yes, is he dead?" "No," he said, "very far from it, he is quite well." And then he told me he had been sent for. The patient was a rich man, he could afford consulta-

tions, and this throat specialist was consulted because the patient had a sort of itching sensation in his nose. The surgeon looked at the nose, and could find nothing wrong, but, by way of doing something, he gave the patient an alkaline lotion to squirt up his nose, after which there was no fever. Undoubtedly, in that case, the alkaline lotion had removed some crusts which had retained pus in some of the cavities communicating with the nose, and so the fever ceased. That is one of a hundred stories I could tell you illustrating the fallacy of thinking that because a fever is intermittent, because the patient's trouble is associated with a high temperature following a rigor and so on, that it is therefore malarial.

I shall assume, in approaching the interpretation of the significance of fever in patients from the tropics that you have eliminated every chance of the possibility of the disease being an ordinary European disease, that you are satisfied the disease is not a common one as opposed to a specifically tropical one. I shall assume that you have gone over the patient's sinuses, his nose, ears, lungs, pleura, liver, kidneys, and do not forget the gall-bladder, nor the appendix, and do not overlook the possibility that you may have a sinus in his kidney, and look for the signs of urethral trouble, and so on. Go over the whole of the organs in the body, one after another. And one thing I would ask you to be very careful about: if you find you cannot account for the disease by ordinary methods of diagnosis, and if you cannot account for it by conjuring up some tropical ailment, think of the possibility of its being malignant disease. I see Sir Thomas Barlow here. He, in my opinion, is well versed in medical wisdom. I also see Mr. Godlee here, who I look upon as well versed in surgical wisdom. And I shall recall a case in which both these gentlemen were concerned, and which conveyed to me an exceedingly useful lesson. The patient consulted me just before I went for my holiday three or four years ago. He had some insignificant pain about one of his ribs on the left side. The spot seemed very tender to the touch, but I could not make head or tail of the diagnosis. I prescribed iodide of potassium—a good thing to do when you are in doubt—and I sent him away. I saw him again a little while afterwards, and he said he was much better. And then the case vanished for the time being from my purview. I went to America and was away a couple of months. When I got back to Liverpool I found a telegram awaiting me there asking me to go to a watering-place in the West of England to see this patient. I went and I found him emaciated, with a long temperature chart beside his bed indicating an afternoon rise of temperature associated with a rigor, followed by a profuse diaphoresis during the night. The case was therefore assumed to be malarial. That diagnosis I had no difficulty in upsetting. But what this disease from which he was suffering was, with its continually recurring temperature, I failed to make out. There were no local symptoms of any sort. He was a reticent, demure sort of man, and he complained of some amount of pain in his back. I thought that possibly he might have renal trouble. I palpated in that region, and examined his urine and tried to eliminate as far as possible every sign of renal trouble before I said I did not know what was the matter. The patient came up to London to be under closer observation, and amongst the people I asked to see him was Mr. Godlee, thinking it might be kidney trouble. We sent for an X-ray expert, and he took a photograph and said he would stake his reputation on the existence of a renal calculus in the patient. But there were no renal symptoms, there was no blood in the urine, no casts and no albumen. I was inclined to doubt the diagnosis, and we asked Sir Thomas Barlow to see him. Sir Thomas said: "I remember when Sir William Jenner was a teacher at this Medical School he used to tell us that when he was very much in doubt about some disease and did not know what it was, he always diagnosed malignant disease." "My opinion is that this case is malignant disease of some sort, but what kind of malignant disease or where it is situated I do not know." The Rontgen rays gave us an indication of the locality of a calculus or trouble of some sort, and Mr. Godlee cut down on the kidney, and found it was malignant, and that inside the kidney there was a concretion of a calcareous character, thus justifying Sir Thomas Barlow's diagnosis, the X-ray man's diagnosis, and perhaps also my own absence of diagnosis.

I remember once, *à propos* of this case and of surgery, being asked to see a lieutenant-colonel of the Indian army who had come home from India invalided with fever. The man was so ill that his

wife came and asked me to see him. I found the colonel in bed with a great temperature chart beside him, of sufficient dimensions to paper his bedroom. Every afternoon there had been duly chronicled a rigor and a temperature of 103° or 104° F., followed by profuse diaphoresis, and the steady record of successive administrations of quinine, up to 30 or 40 gr. a day. This had been going on, not for weeks, but for several months. I examined the patient carefully, and concluded, for various reasons, which I will tell you by-and-bye, that he had no malarial disease, and I told him so. He said: "See that chart, see this periodic fever, see these rigors, it must be malarial fever." So I ventured to differ from him again; but he was so obstinate that he wanted me to examine his blood to see what sort of malignant malaria parasite he had which had refused to respond to the administration of quinine. I said it was useless to do that while he was taking quinine. Protesting, he agreed, and while examining his blood, with negative results as regards malaria, we found it to be positive as regards leucocytosis. I told him that he must have an abscess of the liver and that it should be opened. He refused and wanted to have quinine administered instead. But his wife had considerable power over him, and had more sense than he had, and she insisted upon him having his abdomen opened. I sent for Mr. Godlee, who quickly demonstrated the existence of an abscess in his liver, removed the pus, and converted our military friend from the fallacy of relying upon this daily periodicity as a distinctive mark of malaria.

I will assume you have eliminated all the ordinary causes of fever, and that you are left face to face with an undiagnosed pyrexial condition. Having eliminated all the ordinary causes of fever in non-tropical countries, you assume that the disease is a tropical one. Now put to yourself the question, What tropical disease may it be? Surely there are quite half a dozen tropical diseases associated with fever? The most common is malaria. How shall you recognise malaria? If you go about it in a proper way, and have thorough confidence in your tests for malaria, you will never make a mistake. Believe that. There is no disease so easily and certainly diagnosed as malaria, there is no disease so often overlooked, and no disease so often misdiagnosed, in consequence of want of faith or knowledge and belief in the pathognomonic signs of the disease. There are three infallible tests of malaria. Apply these with faith and courage and you will not make mistakes. The first is periodicity in the phenomena of malaria. The second is what I call the therapeutical test—the action of quinine. And the third and most reliable of all is the examination of the blood by means of the microscope. In every case of malaria, if you apply these tests properly and intelligently and with faith, you will get a positive answer in every case. Unfortunately, as regards periodicity, it is somewhat misleading. Quotidian periodicity is not a property peculiar to malarial fever. You get it in all septic infective conditions; in tuberculosis there is a daily fever, in syphilis even you get it, and in ordinary continued fevers there is an afternoon rise of temperature. Quotidian periodicity is of no value in diagnosing malaria. Tertian or quartian periodicity are absolutely pathognomonic. If you get fever recurring every second or third day you may be quite sure that the patient is suffering from malaria: there is no other pathological condition which gives rise to that type of fever. Put no faith in quotidian periodicity as an indication of malarial disease. Of course you get quotidian malarial fever, but that type of periodicity is not characteristic of malaria, and reliance upon it will mislead rather than help you. You must have something else to go upon. Then try the quinine test. If a patient gets quinine in an adequate dose and it is properly administered, and if the fever does not respond in forty-eight hours to seventy-two hours, that is not a malarial case. Believe that too. I have never seen in the course of my experience—a very considerable one—a malarial case fail to respond to quinine. But you must take care not only that quinine is administered, but that it is in an adequate dose, and that it is absorbed by the patient. It is of no use giving a patient a prescription unless he sends it to the chemist's. It is no good getting a drug from the chemist's unless it is swallowed, and it is no use swallowing it if you do not absorb it. Take care, then, when quinine is given as a test for malaria, that it is absorbed. You know that in malarial disease bilious vomiting is a constant symptom. The stomach is in a state of catarrh and it does not absorb properly, and therefore take care that the quinine which is used as a test for malaria is in an absorbable form. If the

patient is suffering from vomiting and fails to absorb the drug, give it to him hypodermically. Unless the quinine is in such a form and is absorbed so that it is applied to the parasites circulating in the blood, it is of no use as a test. I remember once being asked by a medical man to see his son. His son had been abroad trying to make his fortune fruit-planting somewhere in America. He failed to make his fortune, but he got a bad attack of malaria, and as a consequence he became anæmic, got into bad health, and came home. While at home he had, every now and again, a bad attack of malarial fever, which invariably responded to quinine. But on the particular occasion to which I am now referring the attack was severe, quinine was administered, but the result was not what was anticipated—the fever persisted. In consequence I was asked to see him, the father being under the impression that it was some anomalous form of malaria or tropical fever. I went into all the circumstances of the case, and examined him, becoming convinced that it was malaria. I was astounded at the big doses of quinine which had been taken but had had no effect, so I asked to have a look at the preparation he had been having. A number of quinine tablets were brought to me. I looked at the patient's tongue, saw it was foul, showing that his stomach was undoubtedly in a catarrhal condition, and he had been tumbling these quinine tablets into a stomach which was unable to absorb them. We gave him quinine in solution, and in three or four days the fever had completely disappeared. Remember, then, if you use quinine as a test—and it is an infallible test for malaria—you must take care that the drug is exhibited in such a way that it is absorbed.

The third test which cannot fail, but which is not so easily applied, is the microscopical examination of the blood. Here again, speaking from my own experience, I can say I have never failed in any case of malarial disease in which I had the opportunity of examining the blood microscopically, to find in it the malarial parasite. The presence of the micro-parasite in the blood in malarial disease is an invariable fact—in my experience. Some people have failed to find it; I have never failed to find it in a case of malaria when I have looked for it. But it must be looked for in a proper way and under proper conditions. It will not do to take a patient's blood and put it under the microscope and look through the microscope and move the slide about three or four times and then say there is no malarial parasite. The blood must be carefully prepared, and the individual who examines the blood must also have been carefully prepared by long study of the subject, and he must have practical familiarity with it. For it is no easy matter in the course of a week or two to acquire adequate knowledge of the subject or adequate skill to examine the blood. Anyone who is proposing to practise in tropical countries, anyone who is likely to be brought into contact with tropical disease, must learn to use the microscope for purposes of diagnosis. He must learn the physical characters of the blood, hæmatology, much more than in an immature way; he must know it well and accurately, and he must familiarise himself with the application of the microscope to various blood diseases, particularly malaria. The microscope is of more value in the diagnosis of a tropical disease than is the stethoscope in the diagnosis of lung disease, and were I given two instruments from which to choose for tropical disease, I would discard the stethoscope and retain the microscope, so important is the microscope in tropical disease; it gives you precision, certainty, confidence. I advise any student, before going out to the tropics, to learn his blood examination well, to learn the use of his microscope in the diagnosis of blood disease, to learn to recognise a malaria parasite in all its phases. It is a great comfort to know that you can say positively that a man has malaria, and that if you give him so much quinine he will get well. Do not think it is easy to learn the use of the microscope in the diagnosis of malaria. Sometimes you may search for half an hour before you see the parasite, and unless you are trained you may overlook parasites, or mistake a host of things, such as blood-platelets, blood cells, red cells, masses of dust, etc. It is not enough to have a microscope to be a microscopist.

Thus there is the periodicity test, the therapeutic test, that is, the quinine test, and the microscopical test. If these tests answer in the affirmative you may be sure you are dealing with malaria.

There are subsidiary tests, such as a marked enlargement of the spleen. In malaria there is often a very much enlarged spleen, but so many diseases have enlarged spleens that it is not a

reliable test for malaria. If a man has not an enlarged spleen you may be fairly sure that he has not malaria. But do not be prejudiced too much about spleens. Another interesting point about the diagnosis of the condition is the character of the temperature curve. If you find the temperature suddenly shooting up in an hour or so and then dropping as suddenly in the course of an hour or two to normal and subnormal, it is almost characteristic of malarial fever. Of course there are prolonged malarial fevers, but if you see a temperature chart of that type it is nearly always malaria. Another interesting fact about malarial charts is that in most fevers symptomatic of local disease or general infections the temperature shows an evening rise. In malaria very often the temperature rises before afternoon ; there is a forenoon temperature, a late evening temperature, and an early morning temperature. If you get a case like that you may be fairly sure that it is malaria. Of course you get an evening rise in malaria, but the rises may occur at any part of the twenty-four hours. But that is a subsidiary mark ; you cannot be quite sure.

Perhaps next to malaria the most common disease associated with fever is liver abscess—I mean in the case of patients from the tropics. And that, as I have already indicated, is often mistaken for malaria in consequence of the periodicity and remarkable characters of the fever. In fact, it rarely happens that you find a patient with liver abscess who has not been drugged to the teeth with quinine on the assumption that it is malaria. But, as I have already said, if you find a patient is taking plenty of quinine and yet has not responded to it, it is not malaria. If you would diagnose liver abscess—in many cases it is easy enough—the first thing is to suspect it. Often the first step in diagnosis is to suspect its presence. If you establish the presence of liver abscess, the next thing is to ask the patient if he has ever had dysentery. Liver abscess is almost invariably the sequel of dysentery. Rarely do I meet with a case of liver abscess in which a dysenteric antecedent can be excluded. Then you remark that the fever is quotidian, and you eliminate the possibility of it being malaria by the fact that it has not responded to the big doses of quinine which the patient is sure to have had, and by your not being able to find the malarial parasites in the course of your microscopical examination of the blood, which you should always undertake in a case of liver abscess. And you will find that instead of being deficient in white blood-cells there is distinct leucocytosis. That is very characteristic of liver abscess. In malaria there is always a deficiency of white blood-cells—leucopenia. In liver abscess there is an excess of white blood-cells—in other words there is leucocytosis. Then you proceed to investigate the local symptoms : enlargement of liver, presence or absence of pain, decubitus, and so on. There are half a dozen different symptoms, which no doubt Mr. Godlee has told you of more effectually than I could do, which lead to the conclusion that the patient is suffering from abscess of the liver. But it is very hard, even in the most typical cases, to be absolutely sure of the presence of pus. There is only one test for that, and that is by aspiration, or exploration by the needle. If you find pus by that means you can conclude that it is abscess.

There is, however, one practical point I would like to impress upon you in this matter of exploration of the liver. It must not be done without care. Cases are on record—and there are a considerable number now—in which explorations of the liver have been followed, not by the discovery of pus in the liver, but by hæmorrhage into the peritoneal cavity and the death of the patient. That is a calamity which nowadays should be averted by careful examination of the patient. I will give you a piece of advice founded upon painful experience. If you find an enlarged liver which you suspect to be the seat of an abscess, and if at the same time you find an enlarged spleen, do not aspirate until you have examined the blood. It may be that the patient is suffering from leucocythæmia, and if you aspirate the liver in leucocythæmic patients, the chances are that you may have fatal hæmorrhage from the puncture which you made in the liver. Some time ago a patient came to see me from India, sent home by a medical adviser, with a letter saying she was saturated with malaria, and that she had an enormously enlarged spleen. I examined the patient very carefully, but I could not find an enlarged spleen, though the liver was slightly enlarged. In the course of my examination I discovered a big tumour in the right side of the pelvis. What that might be I could not guess, but I thought probably it had something to do with her ovaries or uterus, and I

sent the case to an obstetric specialist. Before sending her away I made a slide of the blood, with the idea of excluding the possibility of it being malaria. I had no leisure to examine the slide then and I put it in my desk. By-and-bye the obstetrician wrote saying he was very much puzzled; it was certainly not a uterine case, it might be ovarian, but he would like to see me in consultation. So we arranged it. The patient came to my house, and the obstetric surgeon came too, and we concluded that she had some tumour about the pelvis, probably ovarian, and that an exploratory operation ought to be undertaken, and if it were found possible the tumour should be removed. So the patient was sent to a nursing home, an anæsthetist was engaged, and the hour for the operation was arranged. Thinking about the slides I reflected it might be a good thing to examine this slide before the operation came off. I stained it, and was horrified to find that our patient had leucocythæmia, that this huge tumour, which appeared to be ovarian, in her left pelvis, was really a dislocated leucocythæmic spleen. I have no doubt that patient, if she had been operated upon, would have died from hæmorrhage, and her blood would have been upon my head, or rather upon the surgeon's. I mention this as a warning to you not to plunge needles for exploratory purposes without using every possible precaution against so great a calamity as the one I have hinted at.

The next fever I shall speak about as being one that may turn up for diagnosis is Malta or Mediterranean fever. That is a chronic fever with a variety of types of fever. Sometimes it assumes a remittent form, sometimes an undulant form. Sometimes for a week or two there are rises of temperature, then for a week or two absence of high temperature; then another rise and another period of absence of temperature. Perhaps there will be half-a-dozen of those waves of fever following one another. And that is a common type of Malta fever—sometimes a great deal of fever, a temperature of 101° or 102° F., going on for weeks or months, until the patient gets tired of you and you of the patient. A low fever of this sort will puzzle you often in making a diagnosis. You suspect all sorts of things. You think of tuberculosis and percuss the patient's chest two or three times a week, but fail to find any evidence of tuberculosis. You think there may be an abscess or all sorts of out-of-the-way things. You distress yourself and think you have made a mistake, and then you find you have to fall back on the diagnosis of Malta fever. There are certain things which tend to support—I will not say confirm, but support—such a diagnosis. The principal one is the place from which the patient came. This disease is, or was, specially common in Malta until recent sanitary changes had been effected, and in the Mediterranean in general. But it occurs also on the African Littoral, in India, Hong Kong, and various parts of the world. So it may still be met with in patients from other parts of the world. If you find such a fever as this associated with profuse night sweats, and no local disease of any sort, you should suspect Malta fever, more especially if with these night sweats the patient has metastatic, rheumatic-like troubles—to-day one joint swollen, inflamed and tender, then subsiding for two or three days, and by-and-bye another joint becoming swollen, inflamed and tender.

You may, if you are foolish, fall back on serum diagnosis. Serum diagnosis I believe in, theoretically, but I may confess that from the standpoint of practice, and painful experience on my part, I have not the slightest confidence in it. I will tell you what once happened to me in a case of, as I thought, Malta fever. I was asked to see an old gentleman who had been brought to me from the Mediterranean suffering from a type of fever similar to that I have described, a chronic fever of two or three months' standing. The story I was told was that he had been operated upon for middle-ear disease. The operation had been successful, but he was sent out to the Mediterranean to recruit his health. While cruising about there he had fever and landed at Malta, and he remained there suffering with this fever. After a considerable time his friends became anxious about him, and he was brought home to London, and on the supposition that it was probably a tropical disease I was sent for. I considered the story and studied these charts, and came to the conclusion that he was suffering from one of two things: either a recurrence of his middle-ear disease and septic absorption, or else Malta fever. At about that time the serum tests for Malta fever had been brought out. The serum test for typhoid was very

popular, and everyone believed in it at that time. And a serum test for Malta fever had been introduced. I told the friends of the patient and the doctor that the only way of being sure about the presence or absence of Malta fever was to apply this serum test. So we took some of his blood and sent some of it to one laboratory and another specimen to another laboratory. In serum diagnosis I always consult two laboratories. From one laboratory, the following day, I received the reply that the patient was suffering from Malta fever and not from typhoid fever. So I was delighted, for the patient's sake, and having every confidence in scientific diagnosis, like a fool I told the patient's friends he was suffering from Malta fever, as it had been scientifically diagnosed, and it was only a question of a little time and he would get better. Next day I received a report from the other laboratory saying the patient is suffering from typhoid fever; no evidence of Malta fever. So I had to go back and apologise, of course not blaming myself, but blaming the laboratories, and reverse my report, and I thought now he was suffering from recurrence of middle-ear disease. But to be quite certain about the thing I thought I would go to the fountain-head of the serum diagnosis of Malta fever, and so I sent some specimens to Professor Wright, who was then at Netley, and asked him, as the case was an important one, if he would give me a final and reliable opinion on the subject. So, after two or three days, he kindly replied to my request: "I have examined the blood sent me, and have to report that there is no typhoid and no Malta fever." So from three laboratories I had three diametrically opposite verdicts. Therefore I say, in the case of Malta fever at all events, be very cautious about accepting a diagnosis founded upon serum.

I would like to remind you that typhoid fever is very common in the tropics, and sometimes you get patients issuing from the tropics with typhoid fever on them. Sometimes they have recovered from the first attack of typhoid and had a relapse, and it is possible, in these days of fast travelling, to get a patient from the tropics who has contracted typhoid there and the disease is running its course in England. Of course you know the diagnosis of typhoid fever as well as I do, particularly, perhaps, the serum diagnosis.

There is another disease of which I have lately seen several cases, which is supposed never to be seen in this country. I refer to relapsing fever. It is, or was, a common disease in Ireland and some parts of Europe, particularly Russia. It is a very common disease in parts of India, and we now know it is exceedingly common in many parts of Africa. There are apparently two types of relapsing fever although we are not quite sure about it. There is one which we may call the Eurasian type which runs a shorter course than the other type, which we call the African. The Eurasian type of relapsing fever consists of an incubation period of a week or ten days, with a sudden rise of temperature to 103° or 104° F., often associated with jaundice, persisting for a week or so, and then a sudden defervescence in crises. A week of apyrexia and then a first relapse. This first relapse is usually not so severe as the first fever, lasting perhaps a shorter time, and it usually concludes with a crisis of some sort and the disease ends. Rarely you may find there is another relapse, and still more rarely a fourth relapse. That is the Eurasian type. The Asiatic type of relapsing fever is different. There is an incubation period of a week or ten days, and then there is a fever of considerable violence lasting from three days to a week, and then defervescence or crises, a period of apyrexia and then a first relapse. By-and-bye there is a defervescence and then a second relapse, perhaps a fourth, fifth or sixth relapse. One cycle of fever and apyrexia may last ten days or a fortnight, or even three weeks. Therefore if a patient has eight or nine relapses it may be eight or nine fortnights or three weeks before he gets well, and that is, of course, sufficient time to have travelled from any part of Africa to England. For that reason we sometimes see relapsing fever in this country. Only the other day I had a case in the Seamen's Hospital, and a little while ago I had a private patient who had come from Gibraltar. I think these cases are more frequent in this country than is generally supposed. If you come across a case of this kind let me advise you to make a microscopical examination of the blood, not during the period of apyrexia, but while the patient has fever. And if it is a case of relapsing fever and you have skill and patience you are sure to find the characteristic parasite, the spirochæte. Do not forget the possibility of the occurrence of this fever in European

practice, because it may be imported by patients from abroad. I was asked two or three years ago to see a lady who was supposed to be suffering from malarial disease. She had been treated, on that diagnosis, with large doses of quinine. She was pregnant. Probably as a consequence of the large doses of quinine she began to show signs of miscarriage. I was asked as to the propriety, in view of the possibility of this miscarriage, of going on with the quinine. I went to see the patient, and with my usual Scotch caution I refused to give a diagnosis as to the nature of the fever until I had convinced myself as to its exact character. I was told that this lady had spent the previous summer in England and had gone to Gibraltar in August or September, and during her stay there some mosquito had bitten her on the forehead and a big bump had formed there. A few days afterwards she had violent fever, which was associated with the mosquito bite, and the condition was believed to be malarial. It was treated with quinine, and in the course of a week she appeared to recover, thanks, it was thought, to the quinine. To establish convalescence she was sent over to Algeciras, near Gibraltar, but after being there a week the fever recurred. She was brought over from Gibraltar, and the diagnosis of malarial fever was confirmed. She was again dosed with quinine. In due course apparently the quinine cured her and she was well. But after another week she had another attack of what was supposed to be malarial fever. She again recovered, and now the husband was getting tired of it and he thought it was time his wife left there and came to England. After she got to England the fever still continued to recur and she was still treated on the assumption of it being malaria. On hearing this story, and acting on my own experience of the absolute reliability of quinine as to malaria, I refused to accede to the diagnosis of malaria and asked the doctor to send me a specimen of the patient's blood next time the patient had an attack of fever. He did so, and I found that she was suffering from relapsing fever. I think she had had nine recurrences of the disease. I mention this to you to show the importance of not overlooking such a possibility that there may be instances of this African relapsing fever in Europe.

Another tropical fever which you must not overlook is fever associated with the presence of the *Trypanosome Gambiensis* in the blood. I mean sleeping sickness. After a little experience this disease is recognised. The clinical symptoms are almost characteristic, and the microscope confirms it. You suspect the cause in a case which comes from the endemic zone of the trypanosome disease. The patient tells you he is in bad health, that he feels very weak, that he has chronic fever, that he is suffering from headache, and he feels ill and out of sorts. You strip that patient and examine him, especially the skin of his abdomen, thorax, and anus, and the chances are, unless the patient has been under treatment, if the case is European there will be enormous red patches of erythema, sometimes in ill-defined rings, sometimes in great plaques. And you may be sure if a patient exhibits these characters and gives such a history, he is suffering from sleeping sickness or trypanosome disease. To be more certain you examine in detail his lymphatic system, and then you will probably find that he has enlarged glands, especially in the neck, but also in the groin and the axilla and elsewhere. To be quite sure of your diagnosis you examine his blood; make a film of blood somewhat thicker than for malaria, examine it either fresh or preferably stained, and use one-sixth objective and high eyepiece and search for it. If you search for a quarter of an hour or half an hour you will probably come across the characteristic parasite. If you fail to find it in the blood, puncture the lymphatic glands and squeeze a little of the gland juice into the barrel of the needle of the hypodermic syringe, blow it out, and stain it. Examine that microscopically and you will find the trypanosome. Do not give up the idea of trypanosomes being there because you have made four or five examinations; go on examining, especially in times of fever. I am sure that in a case from Africa exhibiting the clinical symptoms I have described, the peculiar fever, the headache, the great erythematous patches on the body with rings or plaques, and enlarged cervical glands, that that person is suffering from trypanosomiasis, and if you examine such cases long enough microscopically you will come across the characteristic parasite. I would utter one warning: make your examinations before the patient has been subjected to specific treatment. I do not think there is anything more remarkable in therapeutics than the power which certain drugs have over this parasite. If you get a

patient suffering from trypanosomiasis you give him one or two injections of antitoxin, and the eruption disappears and the glands become small, and the trypanosomes disappear from the blood. To examine the patient after an injection is, I think, a mistake. If specific treatment has already been commenced when you see the patient, examine him should he have a recurrence of the fever, and then you may come across the trypanosome.

Another recently discovered chronic tropical fever, which comes home often enough to merit attention, and is often overlooked, is kala azar. All tropical fevers at one time were described as malaria, and this particular fever was put down as malarial cachexia; there was a chronic fever, enlarged spleen, enlarged liver, progressive wasting, anæmia, and ultimate death. But within the last few years it has been found, thanks to the work of an Englishman, that it has nothing whatever to do with malaria, although it may occur in malarial countries. By-and-bye, as the disease progresses, there is blood deterioration, hæmorrhages, perhaps diarrhœa, and dysentery, and so forth. Now, if you meet with such a case as that—chronic enlargement of liver and spleen, and an irregular fever—think of the possibility of kala azar and proceed to confute or confirm your diagnosis. This, as a rule, is easily done, first by careful examination of the blood, which, if it be kala azar, is characteristic. There is distinct leucopenia, the number of white corpuscles being reduced very much, perhaps more than in any other disease, there may be only three or four thousand per cubic millimètre. And there is a relatively large mononuclear leucocytosis. The polymorphs are enormously reduced, the mononuclears relatively increased. Study the temperature chart. In most fevers there is only one quotidian rise of temperature, in the afternoon or evening. But often in kala azar you find a double or even a treble rise in the day. There is a spurt of fever and then perhaps a little perspiration, then a decline of the fever, and so on. That is an exceedingly suspicious symptom. Then, in order to confirm your suspicion, proceed to examine the blood, or rather the pulp procured from the liver or from the spleen. Do not do this without the precaution which I have mentioned as to examination of the blood. Do it very carefully, because in these cases fatal hæmorrhage has occurred over and over again in splenic puncture. I have punctured the spleen a good many times and there has always been hæmorrhage. I now choose the liver, because the characteristic parasite of the disease is as abundant in the liver as it is in the spleen, and hæmorrhage from the liver is less likely than from the spleen. To puncture a liver there are certain precautions which should be observed. First as to the interests of the patient. Take care that when you puncture a spleen in this disease you have a binder round the patient's abdomen, and warn him against taking a deep inspiration or expiration at the moment of puncture. Such a thing may cause him to rip up the capsule of his liver and expose him to the risk of rather grave hæmorrhage. Tell him to hold his breath while the puncture is being made. There are two or three points which you need to pay attention to. Use a dry needle; if you use an aspirating or ordinary exploring needle wet, the result of the parasite mixing with a fluid like water, of low specific gravity, is that the parasite changes its characteristic appearance. Do not puncture deeply; there are more parasites in the periphery of the liver than deep in its substance, and there is less danger in puncturing the periphery of the liver. Turn the needle round when you have got it in, so as to break down the liver pulp, and then suck a little of this pulp into your needle, blow it on to a slide, stain it with chromatolin staining, and you will then have no difficulty in recognising the parasite. That is absolutely diagnostic.

There are other forms of tropical fever which I might mention. There is elephantoid fever, for example. It is often forgotten that elephantiasis and elephantoid disease may be associated with a characteristic fever. Some patients get this fever every fortnight, others every month, or every four months or six or eight months. It is quite an irregular recurring fever. But inasmuch as there is a severe rigor, and high temperature and diaphoresis, it is often mistaken for malaria. If you have such a disease associated with lymphangitis or conditions of the leg or scrotum, or great tenderness of the scrotum indicative of lymphangitis of the abdomen, the patient is suffering from elephantoid disease and not from malaria.

There is, lastly, another fever which I would remind you of the possibility of coming across. I have already told you the importance of confessing

you do not know when you are not sure. Whenever you are not sure of a diagnosis, say so, otherwise you are sure to land in trouble sooner or later. It will tell you why. There is a disease in the tropics called leprosy. It is generally associated with a peculiar type of fever—leprotic fever it is sometimes called. This peculiar type of fever sometimes appears for weeks or months, or even years, before the development of the local symptoms. It is quite possible that if you find a patient suffering from a fever and you say it is a trifling fever of no importance, in other words an ephemeral fever, it is possible that by-and-bye that patient may show signs of leprosy. Do not forget that; I have known it occur.

And now, gentlemen, in conclusion there are two or three pieces of advice I would give you. And they are these: First, in approaching the diagnosis of fever in a patient from the tropics, disabuse you minds of the idea that the case is necessarily a tropical one. Remember that the great possibilities are that the case is not a tropical one. Then, if it is a fever, disabuse your mind of the idea that it is necessarily malarial fever. Your first duty is to exclude the possibility of it being malarial fever. Prove whether it is malarial fever, and that you can do with the utmost certainty by paying attention to the character of the periodicity of the fever, by the action of the quinine on it, and by microscopical examination of the blood. I have no confidence in the presence of quotidian periodicity, nor absolute confidence in the occurrence of tertian or quartan periodicity. Pay very little attention to the existence of splenic enlargement. It is always present in malarial disease, but unfortunately for its being a reliable indication of that disease it is present in many other diseases also. Microscopical examination I have great faith in, but you have to take into consideration the personal equation. Do not accept everybody's verdict as to the presence or absence of malarial infection. I see some funny cases occasionally in this connection: patients coming with a diagnosis of malarial fever which is founded upon the microscopist's examination. I ask for the preparation on which the diagnosis was made. Rarely do I get it, and when I do get it, usually the feature in the preparation which was relied upon as an indication of malarial infection is either a piece of dirt, or perhaps an accumulation of blood-platelets, or perhaps a masked cell, or some unusual appearance in the blood itself. Be careful of people who say they understand the use of the microscope and the diagnosis of malaria. Think of the character of the individual who makes the examination. Also beware of serum diagnosis. I have given you to-night only a fraction of my experience. Another thing I would like you to do is always to carry microscope slides with you; this precaution will save you from many a mistake when you visit your patients. With very little trouble you can easily make preparations, and if you have not time to examine each specimen yourself, you can get some of your friends to do it, or send the specimen to one of the clinical research institutes. I have already dwelt on the importance of never giving a positive diagnosis unless you are certain. That is a most important thing. Lastly, remember that we do not yet know all the fevers from the tropics. The history of the past in this matter is only a sort of sample of what the history of the future will be. When I was a medical student we had only one tropical fever known, and that was malaria. Now we have a dozen, all differentiable from one another. What has happened in the past—I will not say how many years—is sure to happen in the future, and very soon, in the next few years I have no doubt, there will be known half a dozen fresh fevers, all at present undifferentiated and unsuspected.

Gentlemen, I must apologise for the somewhat fragmentary style of my address, but I hope I may have given you some hints that in the future may prove of service to you.

February 22nd, 1909.

WITH DR. CAMPBELL THOMSON AT MIDDLESEX HOSPITAL.

Fits after Forty.

Among the methods by which we are in the habit of classifying the incidence of disease, that of grouping according to age occupies a prominent position; and though degeneration of the different organs of the body is frequently disproportionate to their longevity, there are nevertheless broad averages from which useful deductions may fairly be made.

In clinical medicine it is customary to pay special attention to the late "forties," for it is about that time of life that the signs of middle age generally begin definitely to assert themselves. The man is no longer "quite so young as he was." The arteries are less resilient than formerly; the elasticity of the skin is diminished; the "degenerative period" of life has begun. In some the signs show themselves earlier than in others, but whenever they do appear it is plain that they will bring with them dangers which were non-existent during the earlier years of life.

Hypothetically all the tissues of the body ought to degenerate so gradually and so equally that death should finally occur from "old age."

In reality this but seldom happens. Occasionally life flickers out in this way, but as a rule some particular set of functions fails before the rest, and we say that the patient died from heart, kidney or brain failure as the case may be.

On this occasion I propose to consider some of the causes of premature degeneration of the brain-cells and more especially those which give rise to irritation in association with the degeneration.

During the course of cerebral degeneration in a typical old man, the mental faculties slowly fail. Memories for recent events come faint and the perspective of past years is clouded as the period of second childhood approaches. Fits have no place in the course of such natural decay any more than they have in the life of the normal child. But just as the naturally unstable nervous system of the child may be unbalanced by the effects of rickets and other debilitating diseases so that stimuli which would pass unheeded in health may in disease cause convulsions, so in the elderly man may the presence of poisons or variations in nutrition cause that uncontrollable and purposeless discharge which shows itself in the form of an epileptiform convulsion.

Poisons that are powerful nerve irritants may be taken into the body, as in the case of alcohol, strychnine and tetanus, or they may be formed during the course of some disease such as happens in uræmia.

The nerve cells may also be irritated through abrupt changes in their nutrition following embolism or thrombosis, and the irritating substances in such cases may be, at any rate in part, the products of the degenerating cells as is thought to happen in general paralysis of the insane, with which fits are so often associated. Again, gross lesions, such as meningitis, gummata, and other tumours, may all cause convulsions through the general rise of intra-cranial pressure, which their presence creates, quite apart from any localised spasms or Jacksonian convulsions which they may cause by their accidental situation in the motor area of the brain cortex.

We will now approach the subject from the clinical point of view. When called to a middle-aged patient, who states he has had a fit for the first time in his life, the first thing to do is to try to verify this statement. The most careful inquiry must be made regarding the possible occurrence of occasional fits in the past, which may have, perhaps, been separated by intervals of several years. The possibility of the previous occurrence of nocturnal attacks, as shown by waking up with a sore tongue, by finding blood-stains on the pillow, and by the relaxation of sphincters during sleep, the significance of all of which may be unknown to the patient, must also be carefully sought for. Further, the patient must be minutely questioned as to whether he has ever suffered from attacks of "petit mal" in the form of "turns," such as sudden giddiness and momentary losses of consciousness. A patient may have minor epilepsy for years without having a major attack, and quite fail to recognise the nature of his malady until its significance is explained to him.

If it can be established that the patient has been in the habit of having fits previously, either of the major or minor variety, or both, the chances are greatly in favour of the particular attack being another of the same kind, though, of course, the possibility of some new cause being present must naturally not be altogether lost sight of. On the other hand, failure to obtain a history of previous attacks does not enable us to exclude the possibility of "idiopathic" epilepsy with certainty, but it makes the likelihood of its presence improbable because epilepsy comparatively seldom begins in persons over forty.

Are there any signs of a growth in the brain may well be the next question to ask.

If there are there should be general symptoms due to rise of intra-cranial pressure, of which the principal are headache, vomiting, optic neuritis, slow pulse, low temperature, vertigo, and occasionally a generalised convulsion.

Now, general convulsions as an early symptom of a cerebral tumour are not very often met with, and for this reason it is necessary to be on guard lest when they do occur they be diagnosed as "epilepsy." This mistake will not often happen when a case is examined systematically and the greatest safeguard against making it lies in the examination of the optic discs, which should be done as a matter of routine in the case of all patients whom you see for the first time on account of "fits."

So that if with the occurrence of a generalised convulsion we find some of the other symptoms of a rise of pressure we are justified in suspecting the presence of a tumour of the brain, and this suspicion will be confirmed if there are any "localising" signs which will enable us to say where the tumour is. For instance, the occurrence of localised Jacksonian convulsions, quite apart from the generalised convulsions, may point to its situation in the motor area of the cortex, or the signs of pressure on one or more cranial nerves may enable its position to be defined at the base, and so on. Here it may not be out of place to remind you that localised convulsions, usually known as Jacksonian epilepsy, in which the movements begin in a definite group of muscles, proceed along a definite line and are frequently confined to an arm or a leg, are not of themselves pathognomonic of a tumour or of any other lesion of the brain that can be seen either with the naked eye or the microscope. Such convulsions very frequently occur in ordinary epilepsy, and simply indicate that the discharge has not been intense enough to spread over other parts of the cortex, but has spent itself out in passing along the paths leading direct from the cells in which it originated. While, therefore, localised convulsions all have the same significance, inasmuch as they tell us that the discharge which causes the convulsion is taking place in those cortical cells in which the movements of the part couvulsed are represented, they do not tell us the cause of the discharge, and to find this we have to look to the other aspects of the case.

Suppose there are signs to warrant us believing that a tumour is the cause of the generalised convulsion the question as to its nature arises, and to this it is often impossible to give a satisfactory answer.

If it is syphilitic there is always the hope of curing it by medicines, and though many triumphs are recorded it is a hope which, in many cases, is unfortunately not fulfilled, for gummata are frequently accompanied by widespread thickening of the meninges and by changes in the arteries which persist in spite of treatment, or only disappear after the brain has been gravely and permanently injured. Moreover, medicines cannot be relied upon to cure gummata with any degree of certainty, even when these are the sole lesion. Happily they often do, but when it is obvious they are not succeeding, or when there is danger of some important function of the brain being damaged, no precious time should be lost before proceeding to consider the advisability of taking surgical measures, just as would be done were the tumour of another kind.

A great cause for anxiety with regard to a middle aged man who has a convulsion without any obvious reason is the possibility of commencing general paralysis of the insane. Convulsions are common occurrences in this disease, and not infrequently they are found among the earlier symptoms. Sometimes they resemble an apoplectic rather than an epileptic seizure. Careful inquiry and observation will, if not at first, soon reveal signs of mental and physical deterioration, for the fits in this disease generally leave the patient considerably worse than they found him. The chief symptoms to look for on the mental side are undue irritability, elation, or depression, failure of judgment and memory, as perhaps shown by recent errors in business; while on the physical side inequality of pupils of the Argyll-Robertson type, tremors of tongue, lips and hands, hesitancy of speech, and alteration of knee-jerks are among the early signs that are likely to betray the nature of the disease.

Renal disease will not generally cause convulsions till it has reached such a stage as to give no difficulty in diagnosis when there is an opportunity of observing the patient between the attacks. It does, however, sometimes happen that a sufferer from renal disease has periodical attacks of convulsions while he is still able to go about his business, and these, of course, like other convulsions, may at the time give rise to great difficulty in diagnosis to the medical man who has no knowledge of the previous history of the case.

The mere fact that there is some albumen in the urine is not sufficient in itself to warrant a conclusion that the patient is suffering from uræmia, though it may for the time being justify the provisional diagnosis of such. Failure to give sufficient consideration to this point may easily lead to some concurrent affection of the brain being overlooked.

Elderly subjects of chronic alcoholism sometimes have an occasional epileptic fit which does not seem to depend directly upon any failure of the renal functions. Such fits are probably signs that the cerebral cells are undergoing degenerative processes as the result of the steady imbibition of the alcohol. I have assumed for the purposes of this paper that the fits are of organic origin, and so have not discussed the possibilities of hysteria, which must be taken into account in the case of women.

February 22nd, 1909.

SOME UNUSUAL SYMPTOMS IN MYASTHENIA GRAVIS.

A Lecture delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, W.C.

By FREDERICK E. BATTEN, M.D.,
Physician to the Hospital.

MYASTHENIA gravis is a disease which, although not common, yet presents symptoms which are so characteristic that but little difficulty exists in the diagnosis. I propose to-day to show you two cases presenting unusual symptoms, which do not at once suggest the nature of the disease. I would remind you that the first case of the disease was published by Sir Samuel Wilks in 1877, and was described as a case of bulbar paralysis without any pathological lesion. The salient feature of the disease is the rapid exhaustion of voluntary power in muscles after slight exertion. This weakness may at times amount to absolute paralysis, the muscles most commonly affected being those of the eye, palate, and mouth.

As knowledge of the disease increased with the observation of further cases, it was recognised that many cases must be included under the designation "myasthenia gravis," which would not at first sight seem to fit in with the original type.

It was recognised that muscular atrophy was a symptom of the disease, and later it was recognised that cases might present both sensory and mental symptoms.

The typical myasthenic reaction of the muscles in which the muscles rapidly become exhausted to faradic and voluntary stimuli cannot be obtained in every case.

The first case about to be related is unusual, because although there have been symptoms of muscular weakness for some years, yet there is and has been, now four years since the onset, no sign of bulbar paralysis. The second is unusual owing to the long duration and stationary, if not retrogressive, course of the disease. This case has been under observation for sixteen years.

The first case is that of a girl, æt. 25 years, sent to me by Dr. Langridge, of Ilfracombe.

The history as given by him is as follows:

F. B—, æt. 25 years, had been perfectly healthy till the age of twenty-one years, except for childish ailments. She was a strong, healthy child when born, but was brought up by hand. Just before *she was twenty-one years* of age she fell *backwards three or four times whilst rinking.* She hurt her hips and back, but not severely. She felt stiff for about a week and the "bottom of her back-bone was sore." Soon after the fall a rash came out on both legs, front and back, but did not extend above the knees. There was itching, and spots about the size of a pea appeared; they were at first like "bladders" and then formed "scuds," which fell off leaving no scars. She had been wearing a new pair of black stockings, and attributed the rash to some irritating material in these. About six months after her fall—*on July 18th*, 1904, she came under medical observation because of "*weakness in the legs,*" and *because of her having fallen down suddenly in the street and being unable to get home.* She had fallen also a good many times on the stairs. On these occasions she was unable to get up without assistance, or catching hold of something to help her. She also had *great trouble in walking upstairs, and rising from the sitting position*, being obliged to help herself up by pressing on the seat of the chair with her hands, and then she could only get up after several unsuccessful attempts. She felt as if "all power and strength had gone out of her." She was a great strapping girl, well nourished and apparently in good health. Her nervous system was carefully examined, but no evidence of any organic nervous disease could be detected. All her organs and functions were normal, and the condition was regarded as one of hysteria.

After a time she improved considerably, but at Christmas, 1907, she sprained her ankle, and this was followed by a relapse, and she became as bad, or worse than before. She was seen again on October 15th, 1908, and a further examination was made.

She complained of the same symptoms as before. On trying to raise her arms she could get them horizontal with difficulty, but had to drop them again immediately. With an effort she could get them higher, but could not raise them above her head. She had a fair grip, but when carrying anything at all heavy her hands opened and she let it fall. One day she dropped a jug of boiling water and nearly scalded her mother. To do her hair she had to rest her elbows on the table, or hold one elbow with the other hand. The trouble seemed

to be in the movements of the arm rather than of the forearm, the muscles of the shoulder-girdle being at fault.

She stood with her feet somewhat apart, but when asked to do so she could stand perfectly steadily with them together and her eyes shut. She could rise a little on tiptoe. Her walk was decidedly waddling, and she lurched from side to side. *She soon got tired, but after resting could go on walking again fairly well.* She had great difficulty in getting upstairs. When sitting she could extend her leg on her thigh, but when lying in bed she could not, with her legs extended, raise her foot from the bed, again showing that it was the *movements of the proximal segment of the lower extremity that were principally affected, the muscles of the pelvic girdle being at fault.*

She moved her feet freely. In getting into bed she sat on the side of the bed, bent her knees, and, grasping the flexed legs just below the knee in front, lifted them into bed. To get out of bed she first rolled on to her side, then threw the legs over the side of the bed and sat up. If laid flat on the floor on her back, she had the greatest difficulty in getting up, and went through the manœuvres of the pseudo-hypertrophic paralytic child in rising from the floor.

The degree of disability varied, and she volunteered the information that she was always worse about ten days before a monthly period, and began to improve about a week after. Her legs were also worse towards evening.

She complained of a dull, gnawing pain at the back of the neck and in the lumbar region.

She is a well-nourished, healthy-looking girl with good appetite. Legs and thighs look large and well developed, but the upper extremities appear comparatively small and the muscles feel soft and flabby.

As regards *eye symptoms, she often sees double,* but ptosis is only occasionally present. The movements of eyeball are normal, but there is some weakness of the orbicularis palpebrarum. The pupils are ectopic but react normally to light and accommodation.

The *reflexes* are all normal, and the knee-jerk cannot be exhausted by repeated taps.

The *special senses* are normal. She sleeps well and does not suffer from headaches.

The expression of face is rather dull, but all her muscles of expression act naturally. Sensation is perfect to all forms.

The *sphincters* act normally.

As regards *bulbar* symptoms these are entirely absent. She has no trouble in chewing, swallowing, or speaking.

Only a few additions are needed to the above notes. The gait of the patient when tired is very similar to that seen in a pseudo-hypertrophic paralysis. The patient walks with a marked lordosis and tends to waddle, swinging the legs from the hips.

The greatest weakness exhibits itself in the flexions of the thighs on the abdomen.

The electrical examination of the larger muscles does not give the typical myasthenic reaction. Faradic stimulation repeated 200 times does not produce any marked exhaustion in such muscles as the biceps. On the other hand a well-marked myasthenic reaction is obtained in the interossei. The psoas-iliacus group of muscles, which show the greatest weakness, do not lend themselves to electrical examination.

What are the unusual features of the disease in the case?

First, with regard to history. There is the account of the sudden onset of weakness and falling down so that she was unable to get home.

Weakness of the lower extremities is not an unusual symptom, but it is seldom that the weakness comes on so rapidly as to lead to an actual fall. On the other hand a patient with myasthenia gravis may, when out for a walk, get very tired and suddenly collapse.

The most striking feature about the case is the great amount of weakness about the pelvic muscles and especially of the flexors of the thigh on the abdomen. The weakness is to a large extent permanent and is out of proportion to the weakness of the other muscles of the leg.

Such a permanent weakness suggests the possibility of a form of myopathy described by Dr. Thomas Buzzard, in which the psoas and iliacus are the first muscles to be affected. The absence of any atrophy and the presence of myasthenic symptoms are against the diagnosis of myopathy.

The other striking feature of the case is the complete absence of any symptoms pointing to affection of the bulb. The patient can talk well for a length of time without fatigue. There is no difficulty in

mastication or in swallowing of her food. She can whistle and use the orbicularis oris well. In other respects, however, the case is almost typical of the disease.

The absence of what we may call one of the cardinal symptoms of a disease should not allow us at once to exclude a case from a given category.

In paralysis agitans, for instance, it is not uncommon to meet with cases in which there is no tremor, and yet, from the presence of other signs, such as the attitude, the loss of emotional expression and rigidity, cases undoubtedly belong to that group.

Again, in Friedreich's disease the presence of the knee-jerks or of optic atrophy, both unusual symptoms, should not lead us to exclude the possibility of the case belonging to this group although presenting these unusual symptoms.

So in cases of myasthenia gravis (bulbar paralysis without anatomical lesion) the absence of bulbar symptoms should not necessarily exclude the case from this group.

The second case is that of a woman, now æt. 36 years. She attended the National Hospital first in 1893, and complained of difficulty in closing the eyes and general weakness. On examination it was found that her palate was weak. She had a nasal voice and had weakness of the orbicularis palpebrarum. The knee-jerks were normal. The diagnosis of "diphtheritic paralysis" was made, but there was no evidence that she had had diphtheria. She attended the hospital from 1893 to 1897, she then got married, the symptoms remaining much the same.

In 1898 she ceased to attend the hospital until 1905, when she again presented herself complaining of the same symptoms, but her weakness was only marked during her menstrual period. From 1905 till the present time she has been under observation and has had various forms of treatment. During the administration of strychnine she has improved and has remained in good health during the past four years, able to perform all her duties, and though she still exhibits the characteristic features of the disease yet they do not trouble her, and by taking care to rest, especially during her menstrual period, she leads a normal life.

The long duration of the disease in this case is an unusual feature and one which it is most important to recognise when giving a prognosis. The disease is not always progressive and may run a very long course.

It is also of interest from the point of marriage. This patient has been better since marriage. It cannot be said that pregnancy has had any very direct effect on the course of the disease, but in such a disease as myasthenia gravis the possibility must be considered of the fœtus taking on the vicarious function of some absent or diminished glandular secretion. Just as in myxœdema the symptoms of myxœdema tend to disappear during pregnancy owing to the vicarious action of the thyroid of the fœtus, so in myasthenia some similar change may take place. We have, as yet, no definite grounds for this belief, but it is well to bear this in mind when considering all the questions that arise relative to this disease.

Treatment.—Rest is the most important factor in the treatment of patients suffering with myasthenia gravis. Massage and electricity invariably make the patients worse, and should not be ordered. With regard to drugs, some patients have improved on thyroid extract. All the other body glandular extracts have been used, but no marked success has attended their administration.

Strychnine has been used in some cases with benefit, but in others with no benefit—in fact, increase of symptoms has resulted. A quiet, regular life without exertion is that best suited to these patients, and under such conditions life may be prolonged for many years.

In conclusion, the two striking features exhibited by the above cases are first, that myasthenia gravis may exist without any bulbar symptoms, and secondly, that the disease may exist for many years without any increase in the symptoms.

February 22nd, 1909.

VEGETARIANS will derive little satisfaction from a report by Prof. D. McCay. They have been in the habit of citing the Hindu race as an example of the ability of man to abstain from a flesh diet and yet keep in good bodily condition. Professor McCay finds that while the native Bengali maintains nitrogenous equilibrium on his vegetable diet, nevertheless the low nitrogen intake acts deleteriously, reduces the blood protein, and tends to produce degenerative changes, especially in the kidneys. He is physically incapable as compared with the European, and is more easily exhausted. —*Journ. A.M.A.*, vol. lii, No. 4.

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ON THE OCCURRENCE OF PULMONARY COMPLICATIONS AFTER OPERATIONS.

By C. MANSELL MOULLIN, F.R.C.S.,
Senior Surgeon to the London Hospital.

WE have recently had in our wards several patients in whom some pulmonary complication has unexpectedly developed shortly after an operation, and as these complications are often very dangerous and are always very annoying, I thought it might be of interest to try and think out to-day with you why they occur and how they may be avoided. In all the cases to which I am referring the lungs were sound at the time of the operation. There was no reason, so far as we could tell, to anticipate any such occurrence. Of course, if an operation has to be performed under general anæsthesia on a patient who is already suffering from bronchitis, or phthisis, or some other inflammatory affection of the lungs, the inflammation is almost certain to be made worse. That is acknowledged on all sides. Only a few days since we had an unusual example of it in the case of a patient upon whom it was necessary to operate for hernia shortly after he had recovered from an attack of croupous pneumonia involving the right base. That part of the lung apparently had not yet lost its susceptibility. The pneumococcus was still there, and under the influence of the disturbance caused by the anæsthetic the inflammation broke out again, and all the old characteristic symptoms returned. Fortunately the attack was slight and the patient soon recovered. But in the cases to which I am referring now there was no pre-existing condition of this kind. The lungs, so far as we could tell, were perfectly healthy before the operation. Then, within a day or two, sometimes on the very same day, there was a rise of temperature for which the wound did not offer any explanation,

and it was obvious from the patient's breathing and from the physical signs that the lungs were affected. The particular form the inflammatory attack assumed was not the same in all. I have not kept an exact account, but my impression is that, leaving upon one side patients who were already suffering from bronchitis, the most common is lobular pneumonia. Bronchitis comes next. Hypostatic pneumonia is not frequent, except in the aged and cachectic, and, for various reasons connected with the after-treatment of these cases, is not so frequent as it used to be. Pleurisy and croupous pneumonia are comparatively rare. Pulmonary abscess is rarer still. In all the cases I can remember it was secondary to some septic focus in the subphrenic region, usually dependent upon an old chronic gastric ulcer. Pulmonary embolism, to which a great deal of significance is attached abroad, especially in Germany, so far as my experience goes, seldom gives rise to any serious symptoms, unless one of the large branches of the pulmonary artery is involved. It is probably very much more common, in its lighter forms at least, than is usually believed. Many years ago I showed that fatty embolism of the branches of the pulmonary artery was almost always present in cases of simple fracture. No doubt it occurs with equal frequency after operations involving bones, and probably after operations upon other structures also. But I have never known fatty embolism occasion any serious consequences, and if this does not do any harm I do not see why equally minute emboli, occurring after operations, should, always provided that the wound from which they start is an aseptic one. At the same time it must be remembered that each embolus means a damaged portion of the lung, a portion that has lost a good deal of its natural power of resistance; and that if any irritant reaches that spot, either through the air-passages or in any other way, it may easily become a focus of inflammation.

Now it goes without saying that in cases such as these, presenting so much variety, there must be many different causes at work, predisposing and efficient. Some patients, for example, owing to their physical condition are much more liable to complications of all kinds, not merely pulmonary ones, than others. Everything that tends to lower vitality—mental depression, chronic starvation, living in foul air, alcoholism, and above all the cachexia arising from some wasting disease, such as cancer—renders them more than usually susceptible. So does the shock of an operation, from the effect that it has upon the heart. It is not possible to attach too much importance to this. It is the feebleness of the heart's action, combined with restricted freedom of the movements of respiration, that is entirely responsible for the hypostatic congestion of the bases of the lungs in old people, which so soon becomes hypostatic pneumonia, and it is undoubtedly a very important factor in other cases too. Exposure to cold, again, either during or after an operation, acts in much the same way. Fortunately, since our hospital has been reconstructed, the theatres properly warmed, hot-water mattresses provided for the operating tables, and care enjoined that no more of the patient's surface than is absolutely necessary is exposed, this danger has been reduced to a minimum. But it is not always realised that equal care is required after the operation is over, while the patient is still under the influence of the shock. There is good reason to think that a patient who is suffering from shock is for the time being incapable of maintaining the heat of the body at its normal level, in fact is almost like a cold-blooded animal, and that he is largely dependent upon the temperature of the surrounding medium for maintaining his own. Hence the necessity for even more than ordinary care. The position in which the patient is kept after the operation is another of these causes. The head must, of course, be kept low, so long as there is any fear of failure of the heart from shock; but as soon as the pulse rallies the position should be adjusted, so as to give the greatest ease to the respiratory movements. I have never been able to understand why, after so many operations, it should be a fixed rule that the patient should lie flat upon the back in bed, with only a low pillow. The position is a very uncomfortable one for any length of time, causing a backache of which patients often complain more than they do of the operation itself, and is quite unnatural. No one voluntarily sleeps in that attitude. It is, of course, necessary on some occasions, but I am sure that after the majority of operations patients are much more comfortable propped up in bed, or lying upon their side, with as many pillows as they like; and certainly they breathe a great deal better.

But leaving upon one side all of these as general

causes (except where hypostatic congestion is concerned), if you consider for a moment the kind of operation after which pulmonary complications occur most frequently, you will find that the bulk of them fall into one of two great groups—operations about the mouth and throat and operations upon the abdomen. Compared with these, breast operations, in which the patient is under an anæsthetic often for a very long time, and in which an enormous wound is inflicted, sometimes laying bare one side of the patient's chest, are singularly exempt. The explanation is not difficult. Operations upon the mouth and throat are especially liable to be followed by pulmonary complications because of the ease with which, under these conditions, septic material of all kinds can find its way into the air-passages, and the difficulty of clearing the bronchi, as the patient cannot cough; and those upon the abdomen, because of the way in which the respiratory movements are impeded after the operation. Those upon the upper part of the abdomen are the worst for two reasons. Every movement of the diaphragm is very painful, and therefore coughing very difficult; and there is such a free communication between the lymphatics and lacteals below and the pleura above that there is a quite exceptional risk of ascending infection taking place.

Now, as I have said already, it is obvious that there are many factors entering into the production of these complications, and that sometimes one, sometimes another, plays the most important part. But if you look for the immediate cause, there is no doubt that it is the entry into the lungs of infective material in some form or other; and equally undoubtedly this infective material, in the vast majority of instances, finds its way in through the air-passages. The instances in which infection takes place through the lymphatics, as, for example, after operations upon the lower part of the neck, or the upper part of the abdomen, are comparatively rare; and those in which it takes place through the blood-stream are rarer still. The immediate cause is the entry of some infective material. The air-passages form the ordinary route, and all the other agencies are accessory in different degrees.

The infection, therefore, must come from the mouth, nose, throat, or, after vomiting, from the stomach. The mask over the patient's face is always disinfected after each administration, and the air presumably is pure, so that these two can be eliminated at once. Now with regard to the first-named—the mouth—I need not say much. Unless you have paid special attention to the subject, I think you will find it difficult to realise the intensely septic condition of the mouth and teeth in the majority of the patients whom you see operated upon in hospital, and I am sorry to say in not a few of those who are operated upon in private, and who have much less excuse. It is difficult to understand how any of these patients escape infection; and it is certain that except of necessity no operation requiring a general anæsthetic should ever be performed on such unless the mouth has been disinfected beforehand as thoroughly as it can be done. Nor is there any necessity to say much about the nose or throat. The risk of inflammation of the lungs after operations upon the air-passages when there is some form of infective inflammation of the throat or tonsils is too well known. It is not so well known, however, that there is similar danger, though not perhaps in such degree, in cases, for example, of simple follicular tonsillitis. It is admitted that in many of these cases organisms are present, often belonging to very virulent species, though they themselves may not be virulent at the time; and that these organisms may suddenly become exceedingly active, if the conditions are favourable to them. But it is not realised that there is always the possibility in these cases of sudden extension of the inflammation, and it is a wise rule to examine the throat beforehand as thoroughly as the mouth, and if there is any evidence of inflammation, even though it appears to be trivial, to postpone the operation, if it possibly can be done, until the throat is in better condition. The third source that I mentioned is the contents of the stomach after vomiting. This is seldom regarded as of much importance, yet I have seen many instances in which definite attacks of pneumonia have been caused by the inhalation of vomited material through an insensitive larynx, and I have very good reason to suspect that it is an important factor in many more. The greatest possible care is required when patients are sick while under an anæsthetic, or during the period of recovery, to prevent any of the vomited material being sucked down during an inspiration. I am not referring to

cases in which owing to insufficient preparation the stomach is full, and large quantities of semi-digested material are brought up. That may prove fatal at once from asphyxia. The watery fluid and the mucus, often tinged with bile, which is so frequently seen, especially after the administration of ether, is quite enough. It is a foreign body if it gets into the air-passages, and if it is not infective already, it may very easily carry down with it some infective material that it has picked up in its sojourn in a septic mouth.

These are the chief sources whence the infection is derived. The next point to consider is how the infection gets into the air-passages, and the part that is played by the anæsthetic. How far does it deserve the blame? I am afraid it deserves a good deal, though not quite so much as is often laid to its charge. To some extent it cannot help doing harm. The loss, for example, of the protection afforded by the reflex excitability of the larynx is inevitable. One of the great safeguards is gone. It is part of the penalty that must be paid for the production of anæsthesia. The hyperæmia of the mucous membrane of the mouth, larynx and bronchi, and the increased secretion of all the glands that follows are equally inevitable, especially with ether. Vomiting, too, particularly the regurgitation from the stomach of the frothy fluid mixed with mucu sof which I have already spoken, is scarcely to be avoided altogether. But the degree which these unpleasant consequences reach is capable of a great deal of variation; they may be very slight, or very excessive, and these variations are quite under our control. The short administration of gas is very rarely followed by any trouble. With chloroform after-consequences are a little more frequent. With ether, especially when the administration is prolonged, and, as not infrequently happens, more than is actually necessary is given, they are the most common and the most serious of all. I have often noticed how difficult it seems to be for an inexperienced anæsthetist to estimate the quantity of ether that is required when once the patient is thoroughly under the influence of the drug, especially when he has been under some little time. There is such a tendency to continue the administration almost at the same rate as at the start, while really only the minimum is necessary. The result is to cause the secretion of an enormous amount of mucus and saliva. The mouth and nostrils become filled with a frothy mixture, often tinged with blood, which requires to be sponged out at frequent intervals. All the septic material in the mouth and around the teeth is churned up with it. The mouth is propped open with gags so that the patient does not breathe through his nose, and is unable to clear his mouth by swallowing. The larynx, which has lost its sensitiveness, soon becomes filled with the same mixture. All the air that enters the lungs is filtered through the septic froth. Every breath carries the infection deeper and deeper, and at last the septic organisms are carried down into the bronchi, where the mucous membrane is already in a state of intense irritation. It is no wonder that bronchitis or broncho-pneumonia follows. It is not the fault of the ether alone, or even of its excessive quantity. Ether vapour is irritating, and gives rise to hyperæmia and profuse secretion from the mucous lining of all the air-passages, and this may be very serious for a time if the minute bronchi over a large extent of lung are involved. But if the lungs are healthy, and there is no pre-existing bronchitis, this congestion soon subsides, provided there is no infection present. It is the combination of the two, the irritation of the ether vapour and the entrance of the infective material, aided perhaps by the presence of numerous minute emboli which otherwise would be innocuous, that causes such serious results. This is the reason why pulmonary complications are so much more common after operations upon the mouth and throat than after those on any other part of the body; why they are more common when the mouth is foul and septic; why the frequency is proportionate, but not necessarily so, to the duration and depth of the anæsthesia; and why they occur more often after ether than after other anæsthetics; and this will give the clue to the way in which these complications may be avoided.

March 1st, 1909.

Total Avulsion of the Scalp.—Petren has had occasion to treat two women whose hair was caught in machinery and the entire scalp pulled off, and he has collected twenty-four similar cases from the literature. One of his patients died from the injuries, but the other recovered, and the head healed with a number of Thiersch flaps.—*Journ. A. M. A.*, vol. lii, No. 7.

THE CARDIOPATHIES OF THE MENOPAUSE.

By LEONARD WILLIAMS, M.D., M.R.C.P.,
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It is my purpose to-day to direct your attention to a subject of great importance and never-failing interest, of which much too little is heard in this country, namely, the cardio-vascular disturbances which arise at or about the menopause. In France there is a very extensive literature on the subject, and American physicians have not been behindhand in recognising its importance; but in this country we seem so far to have turned a deliberately blind eye to the immense responsibility of the climacteric for the cardio-vascular troubles of later life. Our failure in this respect may be gauged by the fact that the only recognised English classic on the subject * dismisses the whole question in a paragraph of six lines, which begins with these words: "The heart suffers very little at the change of life. . . ." Than which, I need hardly say, nothing could be further from the truth.

In order to obtain a clear idea of these disturbances and their method of production it is necessary to begin by glancing briefly at the vascular phenomena of normal menstruation. At an ordinary menstrual period the uterus and its appendages pass through a phase of increased functional activity, which necessitates an increased blood-supply to these organs. The tributary arteries accordingly dilate, and in obedience to a well-recognised law † there is a compensatory vaso-constriction in some of the other systemic arteries. Were it not for this compensatory vaso-constriction the dilatation in the pelvic area would entail a considerable fall in the general blood-pressure, a state of matters which the medulla, in its extreme jealousy of any restriction of supplies, will never permit. This, then, is what occurs: the pelvic vessels dilate; to compensate for which, there is a constriction in certain other vessels, which, as will appear later, are probably those of the skin. A large quantity of blood is thus forced at high pressure into the uterine capillaries, with the natural result that a hæmorrhage ensues. The amount of this hæmorrhage is considerable. It is estimated at about one-twentieth of the whole of the blood in the body, *i. e.* 200 grms., ʒv–ʒvj.

In the light of these facts it is comparatively easy to understand the otherwise difficult subject of the causation of the sudden suppression of normal menstruation to which all women are more or less liable, an event which is always fraught with unpleasant, and occasionally with alarming consequences. For it is evident that anything which profoundly alters the balance of blood-pressure upon which the menstrual flow depends will have for one of its results the cessation of the flow. An interesting case in point is described by Dr. Francis Hare,* where the inhalation of amyl nitrite immediately checked a normal period; moreover, when a menstruating woman happened to be the victim, the orgies of blood-letting in which our forefathers delighted were always followed by the same result. In the one case the drug dilated the systemic arteries and removed the increased pressure furnished by their constriction: the conditions necessary to the hæmorrhage from the uterine mucosa being thus no longer operative, the hæmorrhage ceased. In the other case the pressure was reduced by the withdrawal of the blood and the remaining force was insufficient to continue the flow. It is the same in the case of the emotional ebullitions, which are known to be potent in the same direction. These ebullitions are accompanied by blushing and other objective vascular phenomena, but also by syncopies, faintings and the like, which, though less obviously vascular in origin, are none the less truly and definitely so. And these nervous disturbances, transitory like the effect of amyl nitrite though they be, are sufficiently profound to alter the distribution of pressure to the point of causing the cessation of the hæmorrhage until the next period. The prolonged absence of the menses which occurs in tuberculosis and other wasting diseases, in typhoid and other acute and subacute conditions, is susceptible of a similar explanation. For in these the subacute and chronic congestions

* 'The Change of Life,' by E. J. Tilt (Churchill, 1870).

† "It follows that every variation in one part is compensated by a simultaneous and contrary variation in another part," Leonard Hill, in 'Schafer,' vol. ii, p. 81.

* "Mechanism of the Therapeutic Action of Nitrite of Amyl," Clinical Journal, August 29th, 1906.

which are of the essence of the diseases, necessarily disturb the rather delicate vascular balance upon which the appearance of the catamenia depends.

In thus briefly recounting the circulatory aspect of the menstrual flow, I must not be understood to suggest that a mere hæmorrhage comprises the whole phenomenon. The fact that menstrual fluid does not normally clot as ordinary blood does, is sufficient to establish a wide difference between the two, and the further fact that menstruation is accompanied by ovulation suffices to invest this particular hæmorrhage with very special characters, the meaning of which will be presently referred to.

This elaborate and complicated process, which commences about the fifteenth year and ceases about the fiftieth, is repeated at regular intervals of about a month during the intervening period. It is thus a rhythmical process dominating and engrossing the whole of that phase of the individual's life. It is essentially a preparation for reproduction, and if impregnation fails to take place, there is what is called an organic disappointment; the blood accumulated in the peri-uterine vessels for the nutrition of the fœtus is discharged, and the preparatory process is immediately recommenced. In the continued absence of fertilisation and of pathological disturbances, this cycle is repeated regularly from month to month until the menopause comes to proclaim that the reproductive period is at an end. The inter-menstrual weeks are not in any true sense weeks of repose from the preparation for reproduction. They are merely weeks of quiet and unobtrusive marshalling of forces which concentrate for the next molimen, and although the marshalling is unaccompanied by any objective sign, its work proceeds as regularly and inexorably as the tides on the sea shore. Menstruation is too often regarded as if it were a spasmodic explosion, but Nature, in her physiological moods, dislikes a hurricane as cordially as she abhors a vacuum.

The elements which are thus gradually marshalled together during the inter-menstrual period are intended for the nutrition of the fœtus, and are therefore, according to Dr. Francis Hare,* mainly carbonaceous in composition. In the absence of impregnation their retention is not only useless but positively harmful, so that menstruation must be regarded as a truly depurative or excretory process. The discovery of new facts in medicine has always had a tendency to obscure existing truths, to the extent even of causing the latter to be so long buried as to necessitate their eventual rediscovery. Something of this kind appears to have happened in connection with the excretory value of the menstrual function. This excretory value has been recognised since the days of Hippocrates, and was insisted upon up to the time of the discovery of ovulation. Then, however, it became so overborne by the importance of what was, after all, but a concomitant and associated act, that it became completely submerged. It is scarcely credible that a writer as recent as Lawson Tait* should have gone so far as to refer to menstruation as "this curious and objectionable phenomenon for which no one has ever yet suggested a useful object." That this object is mainly excretory there can be no manner of doubt. The materials which had been accumulated for the reception and nutrition of the fertilised ovum being no longer required, they are discharged, and with them doubtless various other matters which the economy finds it convenient to expel by this route. In connection with these other matters, it is pertinent to recall that chronic metabolic and excretory disturbances, such as gout, are practically unknown in menstruating women; whereas after the menopause, females are just as liable as males to suffer in this way. It is also well to remember that the excretion of urea is much increased immediately before menstruation, and much diminished thereafter.

But if it be true that the menstrual period is not a mere hæmorrhage, it is equally certain that it is still less a simple excretory process. The phenomenon is, on the contrary, an exceedingly complicated one, to the production of which the internal secretions of various organs would seem materially to contribute. The most important of these organs is, of course, the ovary, a vast deal of knowledge concerning whose functions has been accumulated since the operation of ovariotomy became common. Certain facts have now been established in connection with the internal secretion of the ovary which may be summarised as follows:

* 'The Food Factor in Disease.'

* 'Diseases of the Ovaries.'

(*a*) That it is essential, not only to the development, but also to the preservation of the genitalia.

(*b*) That it causes a vaso-dilation, chiefly of the uterine and peri-uterine vessels, but also to some extent of the vessels in other parts, notably those in nose and larynx. Coryza is a by no means rare accompaniment of normal menstruation.

(*c*) That it exercises an antitoxic effect upon the matters excreted at a normal period. Gautier and Douser * have shown that menstrual discharge contains four times as much iodine as normal blood, and an amount of arsenic equal to the whole quantity normally present in the thyroid gland.

(*d*) That it gives rise to an increase of the output of calcium salts from the body, and—

(*e*) That it has physiological relations with the internal secretions of certain other glands, some of which relations appear to be antagonistic, some complementary, and others of a nature not yet ascertained.

Of these other glands, infinitely the most important is the thyroid. The exact relation between the ovary and the thyroid is still obscure, but we know that in some respects their secretions work in the same direction whilst in others they are antagonistic. The two are in sympathy in being, both of them, essential to the development and preservation of the genitalia. In the matter of the thyroid this is seen in the excellent results of thyroid feeding, not only in cretinism but in all types of infantilism. They are in antagonism in their respective general tendencies. That of the thyroid is definitely katabolic, whilst that of the ovary is as definitely anabolic. The one exception to the rule is provided by their respective attitudes towards the salts of calcium, one of the functions of the thyroid being to fix these salts in the body, while the active ovary aids enormously in their excretion. But the relationship between the two glands does not end here. It is a matter of common observation that the changes which take place both at puberty and the menopause are frequently accompanied by an enlargement of the thyroid gland. It is also a matter of common knowledge that in some women, at any rate, the thyroid enlarges at each menstruation, and in 80 per cent. of women the whole period of pregnancy is characterised by the same phenomenon. Moreover, where there is an insufficiency of the thyroid secretion there is generally scanty menstruation and partial sterility, and a still greater degree of this deficiency produces amenorrhœa and complete sterility. Further, excessive thyroid feeding has been known in several cases to cause pregnant women to abort.

So far as the vascular effects of the two secretions are concerned, though they are both vaso-dilators, each would seem to exercise its effect mainly if not entirely upon the vessels in certain areas. Neither is thus a general vaso-dilator, but each is a vaso-dilator in respect of certain districts. The district under the command of the ovary is, as we have seen, that supplied by the uterine and peri-uterine vessels; the district most conspicuously under the command of the thyroid is that of the skin and subcutaneous tissues. This is suggested by the distribution of the most obvious symptoms of myxœdema, and is supported by the fact that even apart from myxœdema and cretinism, the therapeutic effects of thyroid extract are most noticeable in disturbances of the skin and its appendages. It is probable that the vaso-constriction which occurs at the menstrual period to compensate for the uterine vaso-dilation takes place in the skin; the subjective symptoms point to such a fact. If this be so, the vaso-constriction is probably brought about by inhibition of the thyroid secretion—a probability which is supported by the enlargement of the organ during the period. The transitory nature of the enlargement negatives all idea of hypertrophy. It is almost certainly a retention, and the retention is affected to permit of the constriction of the cutaneous and subcutaneous vessels.

The position of the thymus in this connection would seem to be that of *locum tenens* for the ovary. At puberty, when the ovaries normally become active, it undergoes fatty degeneration, but in the case of animals castrated before puberty it persists for a long period. It would therefore seem that it supplies to the infant economy some principle which in the adult state is furnished by the ovary.

Another gland with which the ovary would seem to have intimate relations is the supra-renal. The relation between the two is suggested by the fact that several cases of sexual precocity have been recorded associated with, and to all appearances caused by, hypertrophy and tumour forma-

* 'Academie de Medecine,' 1901.

tion of the supra-renal capsules. Atrophy of these bodies, on the other hand, is accompanied by very insufficient development of the pubic hair and genital organs. The association would thus appear to be one of physiological antagonism, by which activity in the one gland provokes a corresponding activity in the other, and *vice-versâ*.

From a case reported be Byrom Bramwell* it would seem that the internal secretion of the pancreas is also closely related with the development of the genitalia. He describes a case of infantilism which was cured by the exhibition of pancreatic extract.

To say that there is an intimate connection between the ovary and the mammary glands is to utter a truism, though the exact nature of this connection still remains obscure. The results of treatment with mammary gland extract of certain uterine conditions, more especially menorrhagia and subinvolution, tend to show that the mammæ have an internal secretion which is of importance to the economy. The same may be said of the parotid glands. The relationship of these glands to the ovaries is shown not only by the metastasis which is so liable to occur in mumps, but also by the parotiditis which not infrequently accompanies normal menstruation. These and other facts have induced some observers to try the effect of extract of parotid in the treatment of certain ovarian conditions, and good results are said to have been obtained in neuralgias and inflammations in the pelvic organs.

In this rather chaotic concert of harmony and dissonance it is necessary to notice the sounds produced by yet another instrument, namely, the pituitary body or hypophesis cerebri. There seems to be no doubt that an internal secretion issues from this semi-glandular structure, a secretion which exercises a considerable influence over metabolism and the cardio-vascular system. It was at one time believed to be vicarious in action to the thyroid, but the two substances are certainly antagonistic in one respect, for, whilst the thyroid secretion is a vaso-dilator, the pituitary secretion is almost as powerful a vaso-constrictor as that of the suprarenal gland. Whether the activities of the hypophesis have any direct relation with those of the ovary does not seem to have been ascertained, but its vascular effects render it a factor of very great importance in any consideration affecting the menopause.

The period of life at which menstruation finally ceases is usually quoted as fifty years. The average, even for complete cessation, is probably rather less, and it is quite certain that the phenomena which are liable to characterise the menopause usually make their appearance very much earlier. There may, of course, be no such phenomena. The gradual atrophy of the reproductive organs, which is the essence of the process, may proceed regularly, smoothly and uneventfully to the final consummation. In the conditions imposed by our civilisation, such, however, is rare, and as soon as a woman has passed the age of forty it becomes the part of a careful physician to take into account the possible influence of decrescent ovaries in the causation of any condition for which he may be consulted. From what has gone before it is not difficult to realise that if there is any want of harmony between the involution of the ovaries and that of any of the other organs with which they are so intimately related, the bells of their secretions will be jangled out of tune, and there are liable to ensue symptoms of many kinds in various degrees of severity, which lend to this particular period not only its clinical importance but the interest of its baffling proteanism.

When I began this paper it was my ambitious intention to consider some of these symptoms in terms of the cardio-vascular system and to endeavour so to present them as, in some measure, to facilitate their diagnosis and treatment. The preamble has, however, occupied so much time that I am obliged to devote what remains to a very general survey of this difficult aspect of the question, leaving the details to be considered in a future communication.

We have seen that menstruation is to be regarded, first as a hæmorrhage, secondly as an excretion, and thirdly as the result of an internal secretion, one of whose effects is a partial vaso-dilation which is normally compensated for by a vaso-constriction elsewhere. At the menopause these activities cease, and the cardio-vascular system is called upon to adjust itself to the profound change which is thus affected. I say the cardio-vascular system advisedly, for it is obvious that it is the heart and arteries which will have to bear the

* 'Clinical Studies,' July, 1904.

heaviest burden in the new *régime*. The menstrual hæmorrhage, comparable to a monthly venesection, must mean a monthly relief to the strain on the heart and arteries; a monthly excretion of toxins must purge the blood of impurities, and monthly vaso-motor exercises of dilatation and constriction practised over large areas will maintain, as it were, the arteries in good training. If we assume, as the experience of each one of us entitles us to do, that in a very large percentage of cases the menopause does not occur smoothly, then it is assuredly to the circulatory system that we must look for the earliest and most striking evidence of roughness. And the first question to which I think attention should be directed is that of the blood-pressure. During the reproductive years the systemic blood-pressure is lowest (about 110 mm. Hg.) immediately after a period, and it gradually rises until immediately before the next period, when it reaches from 130 to 150 mm. Hg. With the establishment of the flow the pressure gradually subsides. This process we know to be effected by the internal secretion of the ovary, supplemented and balanced by some other internal secretion or secretions, amongst them probably that of the thyroid. If, now, the vaso-dilative ovarian secretion gradually diminishes to the point of disappearance, while the balancing vaso-constrictive secretion, instead of diminishing, remains constant, it is obvious that a decided increase of general blood-pressure must ensue. Add to this the absence of the pressure-relieving monthly hæmorrhage and the retention of the vaso-constrictor toxins, and we have the conditions eminently favourable to a prolonged and threatening increase of pressure within the arteries.

Those who will take the trouble to measure this pressure, not by the facile but fallacious and futile gauge of the finger, but by the unerring test of the mercurial column, will find that, in effect, a large proportion of the troublesome symptoms of the menopause are accompanied by a very decided rise. Manometric readings of 180 mm. Hg. and upwards are by no means rare, and they obtain for the most part in women who have put on flesh rapidly, and in those whose menopause is effected without the relief afforded by occasional floodings. Dr. Oliver has remarked that what he calls a "wet" menopause is prognostically much more favourable than a "dry" one, an observation which my experience fully supports. In this connection, the following quotation from Dr. Mary Jacobi* is interesting and pertinent:

"We are inclined to think that an occasional menorrhagia followed by several months' intermittence of menstruation is the typical method of establishing the menopause."

But although high blood-pressures are common, more especially in such cases as I have indicated, they are by no means invariable. I have often been surprised to find normal, or but very moderately elevated, readings in women in whom, from their appearance, from the fact of the sudden cessation of the catamenia, and from the nature of their subjective symptoms, I had been led confidently to predict a definitely supernormal pressure. In such cases careful examination of the heart itself has never failed to reveal evidence of dilatation, so that the explanation of these cases would seem to be that the retained toxins had concentrated, so to speak, upon the myocardium, which was consequently bending behind a more or less normal blood-pressure. In a third set of cases, certainly a small minority, which in my experience is drawn from the upper classes, the blood-pressure readings have been quite definitely subnormal. This is difficult to account for, except on the anology of gout, or, rather, goutiness. I used at one time to think that the blood-pressure in this condition was invariably unduly high, but a more extended experience has shown me that this is very far from being the fact. The typically gouty person, the patient, that is, who has receding gums and falling hair, who suffers from acid dyspepsia and a tendency to abdominal obesity, shows as often as not a manometric reading which is subnormal rather than elevated. Why it is that the blood-pressure in this condition should tend to the two extremes in cases which otherwise display so much similarity, I am quite unable to say; nor can I suggest any reason why, in the climacteric woman, it should behave in the same way, except by repeating the well-known fact that her metabolic disturbances at, and after, that period approximate very closely to those which afflict men. She retains toxins which she used formerly to excrete, and of these toxins, sometimes the vaso-dilative will predominate and sometimes the vaso-constrictive.

The moral of this inquiry so far as we have gone would seem to be this: Women at the menopause suffer from a large number of distressing symptoms into any detail respecting which it has

* 'American Journal of Obstetrics,' 1885, p. 601.

been quite impossible for me to enter. Some of these are merely subjective, whilst others are objective, but whether they be the one or the other they are always quite definitely circulatory. The mechanism of these disturbances is still for the most part obscure, but we have in the manometer a method of distinguishing between, at least, two very dissimilar classes, and a means therefore of approaching the treatment of these cases with some degree of intelligence. And the truth is, the ordinary practice of the profession in connection with this matter will bear a little revision. I take down from my shelves a text-book on therapeutics with regard to which I can gratefully testify that it has afforded me invaluable assistance on many occasions, and I turn with eagerness to the section headed "Menopause." Here I find the whole matter comprised in the recommendation of four drugs, namely, bromide of potassium, cannabis indica, valerianate of ammonium, and, as a local application to the head, Eau de Cologne. Whereupon I close the book with a sigh, and bethink me of the saying of the French physician,* to the effect that our therapeutic ignorance with regard to the menopause is undoubtedly responsible for the universal unpopularity of the mother-in-law. In setting ourselves to the study of this question, then, we Æsculapians are undertaking a task, the proper fulfilment of which will cause future generations to rise up and call us blessed, a programme of social reform which is certain to receive the enthusiastic support of both political parties, and one which even the militant suffragette will not venture to relegate to a position of secondary importance.

BIBLIOGRAPHY.

(1) "Organo-Therapy," by Oliver T. Osborne, in Cohen's 'System of Physiologic Therapeutics.'

(2) 'Organo-Therapy,' by H. Batty Shaw (Cassell & Co.).

(3) "La Menopause," by Dr. Vinay, 'Encyclopedie des Aide-Memoires' (Gautier-Villiers).

(4) 'The Food Factor in Disease,' by F. A. a re.

(5) "The Comparative Physiology of Menstruation and the Allied Processes," by Francis H. Marshall, 'International Clinics,' 17th Series, 1907.

(6) "Physiological Phenomena of Menstruation," by Dr. Helen MacMurchy, 'Lancet,' October 5th, 1901.

(7) 'The Menopause,' by Andrew F. Currier, M.D., 1897 (Appleton & Co.).

(8) 'The Menopause and its Disorders,' by A. D. Leith Napier, M.D., F.R.C.S., 1897.

March 1st, 1909.

* B. Bali, 'Leçons sur les Maladies Mentales,' Paris, 1882.

A CASE OF FATAL LYMPHOCYTHÆMIA (LYMPHATIC LEUKÆMIA) IN A BOY AGED SIX YEARS.*

By LEONARD GUTHRIE, M.D., and W. D'ESTE EMERY, M.D.

IT is only within comparatively recent years that lymphocythæmia has emerged from the chaos which reigns amongst the blood diseases of early life, to be recognised as a distinct and separate affection.

For the history and literature of the subject and a complete account of twelve cases, reference may be made to the valuable and instructive paper, "Fatal Lymphocythæmia in Early Life," by Dr. J. Graham Forbes and Dr. F. S. Langmead.†

The disease is characterised by—

(1) The insidious onset of progressive pallor and anæmia.

(2) Spontaneous hæmorrhages from mucous surfaces, purpuric eruptions on the skin.

(3) General enlargement, not invariable and often slight, of the superficial lymph glands.

(4) Enlargement of the spleen, liver, and especially of the kidneys.

The chief distinction between lymphocythæmia and other forms of blood disease is in the excess of lymphocytes, which may be absolute or relative to the total leucocyte count.

The lymphocytes may be either of small or large variety. In Forbes' and Langmead's cases there seems to be no constant relation between the presence of large or small lymphocytes in acute and chronic cases respectively. Dr. Whipham,‡ however, considers that the large lymphocytes are in excess in the acute variety. The leucocytes themselves range in number from some hundreds of thousands, or less than 100,000, to below 10,000 per c.mm.

In some cases there may be even *leucopenia*, especially when a fatal termination of the disease is at hand. But whatever the leucocyte count may

* Delivered before the members of the Medical Society of London.

† 'Proc. Roy. Soc. Med.,' Clinical Section, vol. i, No. 7, May, 1908.

‡ Dr. T. R. C. Whipham, CLINICAL JOURNAL, December 16th, 1908.

be, there is invariably an absolute or relative increase in the number of lymphocytes present.

With lymphocythæmia there is a diminution of red corpuscles to between one and two millions or lower, and a variation of the percentage of hæmoglobin, yielding a colour index above or below the normal.

The clinical course of this disease is marked by variations in the size of the glands, spleen and liver—temporary improvement as regards the degree of anæmia and lymphocytosis. Hæmorrhages of various kinds may be present throughout and mark the approaching termination of the disease, which seems to be invariably fatal.

Pallor and feebleness increase, and death occurs from exhaustion.

The temperature is irregularly raised, but in more prolonged cases, during temporary improvement or shortly before death, falls to normal. There may be albuminuria with casts in the urine, but hæmaturia is rare.

The duration in Forbes' and Langmead's twelve cases was from one week to seven months.

Several varieties have been described by Emerson ('Johns Hopkins' Bulletin,' 1907), namely:

(1) A type like chronic leukæmia, but more acute.

(2) An acute infectious type, simulating acute streptococcal septicæmia in all but the absence of streptococci. In some cases, however, streptococci have been found and cultivated from the knee-joints, glands and blood.

(3) A hæmorrhagic type resembling infective purpura, associated with multiple hæmorrhages and extreme anæmia.

(4) An acute cachectic type marked by languor, loss of flesh, dyspnœa and prostration but few petechiæ, intense pallor and slight jaundice.

(5) A pseudo-scorbutic and pharyngeal form, starting as gangrenous ulceration of the throat, gingivitis or some form of oral sepsis. Finally, some are associated with diarrhœa and vomiting, and with acute nephritis at the onset.

Post-mortem appearances.—The superficial glands are usually slightly enlarged, and also the mesenteric and retro-peritoneal and mediastinal and bronchial glands.

Hæmorrhages are usual in the affected glands and also in the endo- and pericardial surfaces of the heart, the pleura, in the lungs, stomach and intestines, which, however, show no ulceration, though Peyer's patches are invariably swollen. There may be also extensive subdural hæmorrhage.

The liver and spleen show some enlargement, and the former is extremely pale.

The kidneys as a rule are greatly enlarged, even to three or four times their normal size.

They are very pale and extensively mottled with hæmorrhages, which may involve the whole cortex and destroy the definition between cortex and medulla.

The bones show no change. The marrow is seldom increased in amount, and its colour varies from dark to pale red. The thymus is not infrequently greatly enlarged.

"*Microscopical examination* shows extreme hyperplasia of lymphocytes in the adenoid tissues and bone-marrow and lymphocytic invasion of the spleen pulp, the portal canals of the liver, and the interstitial tissues of the heart, pancreas and testicles, and, to an extreme degree, in the kidneys" (Forbes and Langmead).

The following case is typical in most respects of the disease, and possesses certain interesting features of its own.

Case.—Bertie C—, æt. 6 years, was admitted to the Children's Hospital, Paddington Green, May 10th, 1904, on account of increasing pallor and languor with attacks of diarrhœa and vomiting.

His illness was of three months' duration, and began with enlargement of the cervical glands.

On admission he was fairly nourished but very pale, with a waxy, lemon-coloured complexion. The conjunctival and oral and faucial mucous membranes were blanched, but otherwise healthy.

There was no history of epistaxis nor of hæmorrhage of any kind; petechiæ and bruises were absent, but the skin on the front of the legs had a mottled appearance.

Thorax.—Various bruits, obviously hæmic in origin, were heard over the cardiac area. The lungs were healthy.

The veins over the chest, abdomen, and neck were engorged.

Some enlarged shotty glands were present in the neck, axillæ, and groins.

Abdomen.—The liver could be felt $1\frac{1}{2}$ inches below the ribs. The spleen was not palpable.

Urine was normal.

Blood examination (May 26th, 1904):

Red blood-corpuscles . .	625,000.
Leucocytes	2,200.
Lymphocytes . . .	80 per cent.
Polymorph nucleated cells .	20 per cent.

Hæmoglobin 15 per cent.; colour index ·9.

Megaloblasts, hæmoblasts, polychromatophilia and poikilocytosis were absent.

On June 2nd, a week later:

Red blood-corpuscles were 590,000 per c.mm.

Leucocytes . . .	2000
Lymphocytes . . .	78 per cent.
Polymorph nucleated cells.	17 per cent.

The profound anæmia was therefore regarded as not pernicious, but of secondary simple type and probably dependent on intestinal toxæmia.

Under treatment by rectal irrigation and a preparation of hæmoglobin, strychnia and arsenic, rapid improvement took place.

By June 28th:

Red blood-corpuscles had increased from 590,000 per c.mm. to 2,340,000.

Leucocytes were 2000.

Polymorph nucleated cells, 19 per cent.

Lymphocytes, 36 per cent., or nearly normal.

Hæmoglobin was 35 per cent.

Colour index 1·05.

Bacteriological examination of blood was negative.

He had gained strength and seemed better in every way, and was sent to the convalescent home to be kept under similar treatment.

A month later (July 27th) he was re-admitted to hospital on account of rapid wasting, enlargement of the abdomen, abdominal pain, and increase in size of the cervical glands.

Examination of the blood showed a fall of red blood-corpuscles to 1,150,000. Therewere no nucleated cells to be seen now, nor any poikilocytosis. No note was made as to hæmoglobin and colour index.

The cervical glands suppurated on both sides, and on August 11th were operated upon. The pus contained staphylococci.

On September 8th albumen appeared in the urine in considerable quantity for the first time; leucocytes, epithelial cells and a few hyaline and granular casts were also present. Specific gravity was 1005–6.

Thenceforward the patient became increasingly dull, languid, and drowsy. He wasted rapidly, and suffered much from dyspnœa or air hunger, abdominal pain and distension.

Epistaxis occurred on several occasions, but only from the child's habit of picking the nose. There were no hæmorrhages elsewhere nor petechiæ.

The temperature throughout showed a daily rise from normal to 100° or 101° F. It only marked 103° F. on one occasion when the cervical glands were suppurating.

Albuminuria continued, and the patient died exhausted on October 6th, 1904, after an illness of nearly seven months' duration.

Unfortunately no complete examination of the blood was made during the last few weeks of life, but the number of red blood-corpuscles remained at about one and a half millions, and never sank so low as during the first week of stay in the hospital.

The post-mortem examination by Dr. W. d'Este Emery showed that the body was greatly emaciated, and the abdomen was distended and showing signs of decomposition, although the autopsy took place but shortly after death. The muscles did not show signs of wasting, and were of a good colour. The abdominal distension was due mainly to the stomach, which was greatly enlarged and tense. The large intestines were also somewhat distended, but the small were collapsed. The mesenteric glands were greatly enlarged, white, and moderately firm on section. The root of the mesentery was greatly thickened and formed a firm white mass at the back of the abdomen. On section it cut very much like the glands, but was rather firmer. It extended into the retroperitoneal tissues and sent off processes which encircled the aorta. The glands in this region were also greatly enlarged, some being 1½ inch in length. On separating the intestines the kidneys attracted attention at once, as they were of very large size and projected freely into the abdominal cavity. They lay in their normal position, and were readily shelled out.

The kidneys retained their normal shape, all the diameters being uniformly increased, and measured 5¾ by 2¾ inches in each case. Girth at hilum 8 inches. Volume (water displaced), 12 oz. The weight in each case was 12¼ oz. and the organs were absolutely symmetrical. They were of a pale cream colour, almost white, and there were on the outer surface

small pink patches of hæmorrhage, but none in the depth of the organs. On section the pale colour was found not to be quite uniform, for here and there there were regions in which some trace of the original kidney substance could be traced, in the form of medullary substance, the tubules of which were separated from one another by the white material of which the mass was mainly composed. No definite trace of cortical substance could be detected by the naked eye, though in some places the white substance seemed more dense and homogeneous than in others. The capsule stripped with ease, and there was no appearance of infiltration into surrounding tissues.

The suprarenal glands appeared normal.

The liver appeared normal.

The spleen was enlarged to two or three times its normal size, and decidedly firm on section. The cut surface was not uniform in colour, darker and lighter purple areas occurring, but all seemed equally tough.

The pancreas was surrounded by a mass of the growth extending from the mesenteric mass mentioned above, but it was not possible to say whether it was invaded. It seemed abnormally hard.

The intestines appeared normal. The appendix was curiously truncated and felt hard.

To sum up: The kidneys appeared to be uniformly enlarged by a firm (but not hard) white growth which permeated all parts, though not absolutely uniformly, but which did not involve surrounding tissues. The abdominal lymph-glands were converted into masses of similar growth, the glands becoming larger as traced backward, those near the vertebral column being an inch or more long. These enlarged glands formed a chain from the brim of the pelvis to the diaphragm and extended further into the neck. The root of the mesentery was also converted into a thick mass of similar appearance, and sent off-shoots into the retroperitoneal tissue.

The heart was normal; the left lung was largely consolidated, only the edges and apex remaining crepitant—probably partly collapse and partly broncho-pneumonia. There was no sign of growth. The right lung was normal, except for a white speck about as big as a pin's head just under the pleura. The bronchial glands on each side were enlarged and converted into uniform white masses quite similar to those in the abdomen; they were not pigmented. The largest was about 1 inch long. The growth appeared confined to the gland substance and did not infiltrate.

There was a very large gland in the neck which differed from the others in that it showed some black pigmentation round one edge. It was on the left side and deep down just above the thorax. The thymus was present and apparently normal.

The brain was normal except for some œdema of the arachnoid between the convolutions.

Sections were cut from the kidneys, liver, spleen, and the lymphatic glands of two regions. Of these the most interesting changes were in the kidneys and liver.

Kidneys.—The changes may be summed up in one sentence—wide separation of the renal tubules by lymphocytic infiltration, but very little destruction thereof. *The term "lymphocytic infiltration" is used with a certain amount of mental reservation.* The cells are almost devoid of protoplasm, and their nuclei suggest those of a small round-celled sarcoma rather than a true lymphocyte. There is also a very well-marked reticulum between the cells; this is best seen in sections of the kidney, where the cells are widely separated and the whole tissue appears somewhat œdematous. In fact, when the case was first examined, lympho-sarcoma was considered as a possible diagnosis; the resemblance between the lymphatic collections and those seen in some cases of lymphatic leucocythæmia was recognised, but this diagnosis was considered improbable on account of the condition of the blood. The new tissue is deposited apparently uniformly through the kidney, cortex and medulla being apparently equally affected.

At first sight the tubules and glomeruli appear unaffected, and are simply separated from one another by a thick layer of the lymphoid tissue in which they are embedded; in some places this is three or four times the width of one of the tubules. On closer examination it is possible to detect that there has been a small amount of destruction of tissue, fragments of secreting epithelium being found within the lymphocytic mass. It is the good state of preservation of the tubules which constitutes the main difference between this condition and lympho-sarcoma of the kidney; in the latter condition the tubules would be destroyed very soon after they were surrounded by the neoplasm The glomeruli appear normal, except that in some

cases there are a few lymphocytes in the tufts. Bowman's capsule is well preserved, even where it is lying in contact with the lymphocytic infiltration, and the epithelium of the tubules is apparently normal.

Except for the uniform distribution of the new tissue, and the slight destruction of the urinary elements, the kidneys in this case correspond closely to the description given by Forbes and Langmead.

The *liver* is exactly the same as that seen by the authors above-named, and the figure which they give might have been drawn from one of our sections. The liver-cells themselves are not obviously abnormal, but there are masses of lymphoid tissue deposited in the portal canals. These masses are usually angular in outline, being moulded to the surrounding lobules. They vary greatly in size, some being almost as large as a lobule, whilst others are much smaller, perhaps because they are not cut centrally. In some cases a lobule may be seen almost completely surrounded by the material. The capsule is greatly thickened by a deposit of lymphoid tissue exactly similar to that seen in the portal canals; the cells which it contains are more closely packed than those of the kidney.

In lucky sections the characteristic elements of the portal canal (bile-ducts, etc.) may be seen to be quite uninjured, though completely enveloped in the lymphoid tissue.

Spleen.—This was not greatly altered. The lymphocytes seemed to be present in unusually large numbers throughout the section, and in some cases were specially gathered round the blood-vessels. The capsule was not infiltrated.

Lymph-glands.—Here there was a similar diffuse infiltration with lymphocytes, the distinction between cortex and medulla being practically obliterated. The most definite changes, however, were in the sheath, which was greatly thickened with lymphocytic infiltration, which penetrated through it into the surrounding tissues.

The other organs were not examined.

It will be seen that the histological appearances in this case were practically identical with those seen by Drs. Forbes and Langmead in their twelve cases of lymphocythæmia in early life (the microscopic anatomy of one of which was described by Dr. Emery). The main difference arises from the fact that in this case there were no hæmorrhages.

The main features of the case were—

(1) Profound anæmia of non-pernicious type, with red blood count of $\frac{1}{2}$ to $\frac{3}{4}$ million per c.mm., rising incidentally to $2\frac{1}{2}$ millions.

(2) An extreme degree of leucopenia, with a leucocyte count of only 2000–2200, of which 78·80 per cent. were small lymphocytes.

As already mentioned a sudden fall of leucocytes and lymphocytes is not uncommon in the course of the illness, and usually occurs shortly before death. In three of Forbes' and Langmead's cases the total number of leucocytes fell to 5000, 3500, and 3000 respectively, when death was imminent. But in this case marked leucopenia was present four months before death took place.

The remarkable rally in which the red blood corpuscles rose from $\frac{1}{2}$ to $2\frac{1}{3}$ millions in the course of a few weeks, and the percentage of lymphocytes became nearly normal, the leucopenia still persisting, is also worthy of attention.

Another point of interest is the absence of spontaneous hæmorrhages throughout the illness. In order to account for it one can only suppose that the walls of the blood-vessels remained healthy.

Finally, the enormous size of the kidneys, each of which weighed $12\frac{1}{4}$ ozs., the increase in size being due to their being densely packed by myriads of lymphocytes, is unique in the records of such cases.

The origin of lymphocythæmia still remains obscure. Robert Hutchison* suggests that the activity of adenoid tissues in early life and their consequent liability to pathological changes are responsible, and that over-growth of the marrow-cells, which are the supposed progenitors of lymphocytes and myelocytes, may be the cause.

Many favour the view that the bone-marrow is at fault and that the process is essentially malignant or sarcomatous in nature; the preponderance of the large variety of lymphocytes in some cases seems to lend support to a myelogenous origin of the disease. On the other hand, the enlargement of the glands in general and of the spleen and thymus and the preponderance of small lymphocytes support their origin in the lymphatic glands and tissues. Yet intermediate forms of cells are sometimes present, and it is impossible to deter-

* R. Hutchison, 'Lancet,' vol. i, p. 1331, 1904.

mine whether they have a myelogenous or lymphatic origin.

In early childhood the functions of the marrow and adenoid tissues are not differentiated as in later life.

Such changes as have been found in the marrow are not sufficiently distinct to settle the matter. The marrow may be pale or lighter than normal, and show the invasion of lymphocytes seen in other structures, but sometimes it is dark or bright red in colour.

Microbic infection of the blood as the cause of lymphocythæmia finds many supporters and not a few opponents. Streptococci have been found after death in the blood, spleen, and marrow, and occasionally during life in the blood. The onset with various kinds of oral sepsis in some cases, the rapid course and pyrexia which attend the disease, seem to prove a microbic or toxic agency. Yet the presence of organisms may only indicate an intercurrent or terminal infection. The rise in temperature may depend, as *Treadgold** thinks, on the action of degenerated leucocytes on the heat centres. In our own case bacteriological examination of the blood proved negative. The pus from the suppurating cervical glands was staphylococcal.

On the whole it seems most probable that a microbic or toxic infection of the blood may set up changes in both lymphatic glands and marrow, and give rise to excessive formation of lymphocytes, which are deposited in enormous numbers in the various organs and tissues.

Whether such a reaction to an irritant can be regarded as malignant or sarcomatous in nature must at present be left undetermined.

As we have stated, the cells in our own case suggest those of a small round-celled sarcoma rather than a true lymphocyte. Yet the absence of extensive destruction of the renal tubules, although invested by densely packed cells, points to simple hyperplasia and not to malignant growth. It has been suggested that excessive lymphocytosis is a protective action against micro-organisms or toxins, which fails. Such teleological explanations, however, are hardly ever satisfactory.

In conclusion, although lymphocythæmia so called is undoubtedly a definite and separate disease as shown by the post-mortem appearances, it is doubtful whether it can be diagnosed with certainty by examination of the blood during life. Excess of lymphocytes is held to be pathognomonic of the disease, but it must be remembered that in early childhood lymphocytosis may be present in many other morbid conditions of the blood, all of which are protean in character, so far as the results of examination of the blood are concerned.

As regards terminology, it seems paradoxical to describe a case such as the present as one of lymphocythæmia, when the total leucocytic count only amounted to 2000 per c.mm. Yet, though not in excess in the blood, the lymphocytes—if, indeed, they be lymphocytes—were deposited in incalculable numbers in the tissues and organs. No doubt in time a more appropriate name will be found for this particularly fatal affection.

Dr. PARKES WEBER said that, in the present state of knowledge, Dr. Guthrie's case could not be regarded as one of lymphocythæmia; there was no blood-picture (quantitative or qualitative) of lymphocythæmia present in it at all. The kidneys exhibited a characteristic infiltration or permeation with growth made up of lymphocyte-like cells, as Dr. Guthrie had described. That condition was already known when, twelve years ago, he himself described* before the Pathological Society the case of a child with similar kidneys. In addition, in his own case, part of the intestinal wall was affected, and had become enormously thickened with the same kind of diffuse growth. Such cases were examples of lympho-sarcomatous permeation or lympho-sarcomatosis; by lympho-sarcomatosis he did not imply that there was a growth burrowing its way into the arteries etc. At any rate there was very little indeed in the present case to justify the term "lymphocythæmia." There were cases of lymphocythæmia in which there was leucopenia, especially from terminal infections, but there was no terminal infection in the present case. Neither did the differential blood count of the white cells support the diagnosis of lymphocythæmia.

Dr. D'ESTE EMERY, in reply to Dr. Parkes Weber said that if Dr. Weber applied the same criterion in the present case to that which he applied with regard to mediastinal tumours he would find that

* 'Quart. Journ. of Med.,' 1908, vol. i, p. 239.

* F. P Weber, "Diffuse Symmetrical Lympho-sarcomatous Infiltration of the Kidneys of a Child," 'Path. Soc. Trans. Lond.,' 1906, vol. xlvii, p. 117.

it was typical leucocythæmia or lymphocythæmia as described by Forbes and Langmead. In discussing Dr. Wall's paper, Dr. Weber said the criterion to apply was the structure, and with that he (Dr. Emery) agreed. The structure of Hodgkin's disease as a characteristic of the disease could not be diagnosed with certainty until the sections had been cut, and until they had been examined by somebody who was familiar with the condition. In the present case the microscopical anatomy of the tissues, except the bone-marrow, which was not examined, was exactly the same as that of Forbes' and Langmead's cases. The fact that the blood count did not confirm that was another matter. Their ideas of lymphatic leucocythæmia or leukæmia had undergone considerable alteration in the last years, and his own certainly had. Ewing said one could not diagnose the condition with certainty unless there were less than 150,000 leucocytes, of which 90 per cent. must be lymphocytes. Of course there was no difficulty in diagnosis when that degree was present, but there were gradations. He saw a case not long ago in which there were never more than 10,000, and he searched through several films and found nothing but lymphocytes. It must be admitted at present that numbers were not essential to the diagnosis of leucocythæmia. But now there were the typical post-mortem appearances in the kidney and also in the spleen and in the glands, as in the twelve cases of Forbes and Langmead, which contribution must be looked upon as giving a very vivid description of a certain type of the disease. He had described one of their cases, and he had no doubt that the present case was of the same kind. The notes regarding lympho-sarcoma which Dr. Guthrie had just read were his (Dr. Emery's), written four years ago. But when he saw the sections he decided that the term "lympho-sarcoma" could not be applied, otherwise one's ideas of a malignant tumour must be altered, for in this condition the organ was packed from end to end with newly-formed lymphoid tissue, but there was no destruction of the organ. Lympho-sarcoma proper destroyed tissues more quickly than almost any other form of malignant growth.

Dr. Weber, in explanation of his previous remarks, said he agreed with Dr. Emery's principles. He admitted that lymphocythæmia, like lymphadenoma, could be distinguished by its histology, but in lymphocythæmia the histological characteristic was a morphological change in the blood, which had not been observed in Dr. Guthrie's case. If Dr. Emery objected to the term "lymphosarcoma" or "lymphosarcomatosis" being applied to cases like Dr. Guthrie's case, on the ground that the kidneys were infiltrated without the renal tissue being destroyed by the lymphocyte-like cells of the growth, then he (Dr. Weber) would suggest the use of the term "lymphocytoma" or "lymphocytomatosis" for the cases in question. Diffuse lymphocytomatosis would simply imply diffuse infiltration by a growth consisting of lymphocyte-like cells.

March 1st, 1909.

Dr. Jabez N. Jackson considered that the term "pericolitis" had been generally vaguely used to describe adhesions about the colon, and at most times of an unknown origin. He believed that there was a form of pericolitis which had a distinct pathological identity, and was characterised by a distinct clinical picture. This pericolitis was characterised by the formation of an apparently entirely new layer of distinctly vascularised peritoneum. This membrane served to bind the ascending colon close to the posterior abdominal wall, and the longitudinal strands tended to produce a shortening and often a marked angulation of the colon. Sometimes this membrane passed across the ascending colon and up on to the transverse colon, which was then drawn downward and to the right until the transverse colon paralleled the ascending colon and distinct angulation was produced at the hepatic flexure above and below where the transverse colon turned back. With this latter condition the stomach was always drawn down and a mechanical gastroptosis was produced. The general effect of this membrane was a mechanical interference with the free peristalsis of the colon, also the stomach, thus producing stagnation, distension, pain, flatulency, and a train of nervous symptoms distinctly neurasthenic in character. This condition was believed to be of very frequent occurrence, and must have been observed by all surgeons of experience, but thus far had not been adequately described. He had found that this membrane could be dissected off *in toto*, and when this was done the mobility of the colon was restored.—*Medical Record*, vol. lxxv, No. 7.

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A CLINICAL LECTURE ON ANEURYSM.

Delivered at St. Bartholomew's Hospital.

By ANTHONY A. BOWLBY, C.M.G., F.R.C.S., Surgeon to the Hospital.

GENTLEMEN,—Here is a specimen of an aneurysm of the popliteal artery. You will see that it is of fusiform shape and that the whole of the popliteal artery and the lower portion of the femoral artery are involved. You will also see that the aneurysm is full of clot. Nine years ago I saw, with Sir Thomas Smith, a gentleman æt. 73 years, who had quite suddenly been seized with gangrene of his leg. It involved the whole of the leg as far as the popliteal space. When we came to examine him with the view of finding a cause for this gangrene we ascertained that he had an aneurysm in his popliteal space, which he had been quite ignorant of up to that time. The clotting of the blood in that aneurysm had resulted in gangrene of his leg. Under those circumstances there was only one thing to be done, namely, to perform amputation. This was carried out by Sir Thomas Smith, at the junction of the middle and lower thirds of the thigh, and the patient made a perfectly satisfactory recovery. On examination the popliteal artery showed very much the condition which you see here, namely, an aneurysmal sac filled with clot and blocking up the circulation in the whole of the vessel. When we examined this patient we also investigated the condition of his other blood-vessels, and we found at the time of the operation that he had an aneurysm in his other popliteal space, of which he was quite unconscious. We had to consider whether it was advisable to do an operation on this aneurysm, and decided that as he was an old man, æt. 73 years, and as he was about to lose one leg, he was not likely to lead

a very active life and that it was not advisable to do anything to this other aneurysm; so we did not even inform him of its existence. You see, then, the position of affairs was that he had had spontaneous gangrene of one leg due to unrecognised aneurysm of the popliteal space, and that this gangrene had been treated by amputation, and that he recovered. Now, in regard to the other leg, what was the expectation of the course which this second aneurysm would pursue? No one could say that the same thing might not happen in it as happened to the first aneurysm, though the more common thing is for an aneurysm to increase in size. This aneurysm did not change at all. The patient continued unconscious of its existence, and led as active a life as a man with only one leg and at 73 years of age was likely to lead, and for nine years the aneurysm did not alter. Two months ago, and he was then 82, exactly the same sequence of events occurred in the remaining limb as that which had occurred on the right side, namely, coagulation of the blood in the aneurysmal sac, gangrene of the leg, amputation of the thigh, and still, fortunately, the same result as in the first amputation—complete recovery. So at the age of 82 years both his lower limbs have been removed. The case is interesting from several points of view. I want to first of all point out that this case of aneurysm illustrates a fact which is hardly sufficiently appreciated, namely, that in a certain number of cases aneurysms neither get well nor increase, but remain unaltered. And that indicates that you should not be in too great a hurry to decide at once, as soon as you find an aneurysm, even if it is favourably situated for operation, that operation is the proper treatment for it.

Let me tell you one or two other cases illustrating the same fact. A doctor who was 63 years of age, who had had a very hard life, discovered that he had an aneurysm in his popliteal space. He was greatly concerned at this discovery and immediately concluded that he must probably submit to an operation for ligature of the vessel. When he came to be examined, we found that he had certainly a dilated heart, and to his surprise there was an aneurysm in the other popliteal space. He was senior partner in a practice in which there were two other partners, and he had already made up his mind that he was going to retire from practice in another year. We persuaded him that the best thing to do was to retire at once and take up an easy mode of life. He asked, in some surprise, whether it was not advised that he should have an operation, and we replied that it would be better to see what was the result of his comparative inactivity. These aneurysms remained absolutely quiescent. He did not walk much, but drove a good deal, and never had any further trouble from his aneurysms at all. He lived for several years after that, and ultimately died from gradually increasing heart mischief.

Thirty years ago a patient came to this hospital with symptoms, as it was believed, of aneurysm of the aorta. She was seen by one of the physicians, who diagnosed that she had aneurysm of the aorta. She remained under observation at intervals for nearly twenty years, and during the whole of that time this aneurysm did not give any further definite physical signs. At the end of that period of twenty years she was in the hospital for chronic bronchitis and dilated heart, and of these she died. Post-mortem examination showed that she had, as had been diagnosed, aneurysm of the descending aorta over the greater part of the length of it, and it was probable that this had not materially changed during those twenty years.

One more case. A patient, who was at that time in an infirmary, was sent up to see me here at the age of 73. He had an aneurysm in his iliac region, on the left side, above Poupart's ligament, as large as a small hen's egg. He said he thought this had been there nearly two years. He was a feeble old man, and being in the workhouse infirmary it was known that he would be doing no more hard labour. I advised, under the circumstances, that he should not be taken in and operated upon, but that he should be kept under observation, and not allowed to do work of any kind. That man, for seven years afterwards, used to come up to see me at the hospital. On many occasions he went to the College of Surgeons to the examinations, and I cannot tell you how many times he was advised to have his common iliac tied by candidates. But at any rate he continued to do without operation, and at the end of seven years I had a letter from the doctor at the infirmary to say that at last he had died. That aneurysm practically did not increase at all.

I mention these cases because I do not think it is sufficiently appreciated that in old people who

do not have laborious work aneuryms sometimes do not increase. I suppose that is due, first of all, to the fact that in very many of these patients there is not the same powerful drive of the blood that there is in a comparatively young person with a muscular heart. You know that in all aneurysms there is a tendency for the artery to be thickened so as to resist the strain which the heart is putting upon it. If you examine any vessel which is the seat of aneurysm you always find that before there is that thinning away of the coats of the vessel which may precede a rupture there is a thickening and matting together of the coats; that the middle coat and the external coat and the sheath all become thickened by the formation of fibrous tissue. These are attempts on the part of Nature to repair the over-stretching of the vessel, and in a certain number of cases Nature is successful and sufficiently strengthens the vessel to prevent it being further materially dilated. The conclusion which I think we must draw from that is, that in old people with aneurysm which has been recently discovered it is not well to be in a great hurry to advise operation; that we should make quite sure that the aneurysm is increasing, in spite of ordinary care, such as rest and the avoidance of hard labour.

The next thing to consider is, What is there in an aneurysm except spontaneous coagulation which can give rise to sudden gangrene of a limb? Here is a portion of the leg and the popliteal space from a patient who was admitted into Darker Ward nearly a year ago; it is a section taken through the tissues behind the tibia, and you will see a large mass of clot, in the midst of which it is extremely difficult to trace the vessel. This was from a man aged only 37, who suffered from an aneurysm of the popliteal space, which ran a different course from that which I have described in the old man. And I ask you to notice the difference between the two. This patient I am speaking of now was comparatively young, only 37 years of age, and he was leading an active muscular life, and was in active work. As a result of the heart driving the blood forcibly this aneurysm increased in size, and it became more and more sacculated or pouched, so that the sac began to give way and the blood began to percolate through it into the surrounding parts. Up to this time apparently the patient had taken no particular care of himself at all, but now that his leg was beginning to swell and the popliteal space was painful, he sought advice. But he was rather too late in seeking it, for by the time he came into the hospital we found that his leg was already threatening to become gangrenous, and there was considerable extravasation of blood, extending from the bottom of Hunter's canal into the calf. And although I tied the artery in Hunter's canal as soon as he came in, the extravasation had gone too far. There was so much interference with the circulation of the limb that it was already gangrenous, and amputation had to be performed. But although in this case of rupture of an aneurysm the gangrene came on gradually, you must understand that in a certain number of such cases it comes on suddenly. Some years ago an old gentleman, æt. 73 years, himself a doctor, was suddenly taken with intense pain in his knee and leg. The leg swelled and became excessively painful, and within forty-eight hours it was gangrenous. He had had aneurysm of his popliteal space which had become thin and burst, and having burst into the popliteal space the extravasated blood so interfered with the circulation that it brought about death of the limb in a comparatively few hours. So the gangrene of both legs in the first case which I have alluded to was due to a different cause from that which was operating in these others. In the case of the old gentleman who lost both his legs the death of the limbs was due to the coagulation of the blood in the aneurysmal sac, and in these other cases it was the result of the sac giving way and the blood becoming extravasated and forming a large mass in the cellular tissue, thus compressing the vein and the artery and the small arteries as well. Now, supposing for a moment that the patient who lost both his limbs had had his artery tied, the only effect of that would have been to produce that very condition of coagulation of the blood in the aneurysmal sac which, even under the conditions which existed, brought about the gangrene. So, by not tying the vessel at the time when we first discovered his second aneurysm, we gave him the use of his limb for nine years. On the other hand, in the patient in Darker Ward, the operation of ligature of the main vessel would have prevented gangrene. This man lost his leg, because he came in after the aneurysm had burst and after the clot had compressed the vessels; in all probability he would have saved his leg if he had had his artery tied soon enough. You see the great difference in the cases is that one man was thirty-seven and the other was seventy-three. In the man who was thirty-seven there was a reasonable prospect that the other vessels were sufficiently elastic and healthy to enlarge and carry on the

circulation. In the old man the other vessels were rigid, and were too inelastic to allow of their carrying on that function, their muscular coat was no longer what it used to be, his vessels were much more like rigid tubes, and when a demand was suddenly made for them to carry on extra circulation they were unable to respond to it. But in the young patient, if the artery had been tied sooner there is every reason to suppose that the collateral circulation would have been adequate and he would have recovered.

There is another cause, apart from aneurysm, of sudden blocking of vessels, and that is embolism, and by it a previously perfectly healthy artery may be suddenly occluded. The embolus or foreign body which blocks it almost invariably comes from a diseased heart. Here are the toes of a woman who was admitted into the hospital under these circumstances. Some ten days previously to admission she had been confined, and about the tenth day after that event, on getting up out of bed she suddenly had an intense pain in her thigh and groin. At first her foot was burning and tingling, and subsequently it became numb and cold. Afterwards the toes became so numb that she could not feel them. When she came into the hospital her toes were already beginning to be gangrenous. Examination of the thigh showed there was no pulsation in the femoral artery below Poupart's ligament. Above that point it could be ascertained by pressure that there was a healthy vessel. The pain was of sudden onset, and there was arrest of pulsation at a certain point. That all pointed to the fact that the patient had had a lodgment of an embolus, which had blocked the main artery and had so interfered with it that the circulation of the limb could not be completely carried on. She was young and otherwise healthy, and the greater part of the limb could be nourished by collateral circulation, so that she lost only her toes.

Here is a picture from a patient who also suffered from embolism. He was a man about sixty years of age, an unsound man with much disease of the heart, and the story was that whilst he was employed in cleaning boots he suddenly had intense pain in his arm. He dropped whatever he was holding and the pain was excruciating. At first the hand felt very hot and burning and at the same time numb. After that it gradually became cold, and ultimately. he completely lost all sensation in the arm, and before very long it became gangrenous. Amputation was performed just above the elbow, and as far as the arm and the operation were concerned he did well. But all this was too much for his already diseased heart, and he rapidly became worse and died. Post-mortem examination showed that his main brachial artery had been blocked at the bifurcation of the radial and ulnar, and that below that some of the small arteries had also been blocked by clot. These are two cases in which the gangrene was due to embolism, and in them there was no pre-existing arterial disease at all. The origin of the gangrene was mechanical; it was due to the vessels being suddenly occluded. But occasionally the embolus may be more than a mere portion of fibrin or blood-clot, for it may contain micro-organisms, and therefore be what is known as a "septic embolus." It may come from a heart which is affected with ulcerative endocarditis. Here are two pictures from the case of a young woman, æt. 19 years, who came here on account of pain in her calf. This pain in the calf was apparently of sudden onset, and nothing was felt at the time she was first seen in the casualty department. But before many days she came back and said the calf had become more painful and that it had swollen. On examination it was found that there was a pulsating swelling, and she said that in addition to that she had a swelling in her arm. In her forearm there was found another pulsating swelling. These were evidently aneurysms. She was admitted into the hospital, and before very long another aneurysm was felt in her iliac region. So you see she had three aneurysms, and before she had been in hospital many days she was suddenly taken with hemiplegia and brain complications and ultimately died. Soon after her admission it was found that she had morbus cordis, and the general conditions pointed to a septic state of her heart, so that a diagnosis of ulcerative endocarditis was made. The heart from this patient showed, after death, typical ulcerative endocarditis, and the emboli derived from the ulcerating valves were septic. Not only, therefore, did they plug the artery, but they caused, at the seat of plugging, a septic inflammation and ulceration of the vessel. Here you will see a portion of the brachial, radial and ulnar arteries, and there are small calcareous portions of clot in them, and here is a hole in the artery showing where it has been ulcerated through. So in this case the septic embolus has caused exudation of blood through the hole in the artery. When you come across a very young patient with an aneurysm you should always suspect that that aneurysm is embolic in its origin. Aneurysms in people under twenty-five years of age are seldom due to atheroma, which is essentially a degenerative disease, occurring in advanced middle age or in old age, or in young people whose arteries are affected by syphilis. Where you find there has been lodgment of a septic embolus in a young person, be on the look-out for ulcerative endocarditis, and you must remember that what has happened once may happen again, and that this embolus which you find may not be the only one, but that, as in the case of this girl, æt. 19 years, there may be multiple emboli affecting many vessels and causing several aneurysms in the same patient.

March 8th, 1909.

OCCULT "PRIMARY CARDIAC HYPERTROPHY."

Being a lecture delivered at the Brompton Hospital for Consumption and Diseases of the Chest.

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THIS title for a clinical lecture has been chosen, not because of its familiarity nor because of its popularity, but simply because physiological experiment and clinical experience have shown that the unaided finger is not so well able to estimate blood-pressure as is the sphygmomanometer, so that cases of cardiac hypertrophy are the more readily missed. It is a lamentable fact that the profession at large hesitates to make use of instruments of precision in the difficult problems of diagnosis. However, facts as they are must be accepted, and it must be acknowledged that the history of medicine shows a great disinclination on the part of the profession to confess that the *tactus eruditus* is not to be relied upon. A glance at contemporary medical literature will show the hesitation exhibited when the stethoscope and clinical thermometer were introduced; even to this day our French colleagues prefer, above all nations, to practice, with its obvious drawbacks, immediate auscultation of the chest, and still a few are found who maintain that the hand can detect a rise of temperature of the body.

If we can but persuade ourselves to open our eyes to the defects of older views, then we should be more than half-way to reform. Old habits of thought are difficult to change, and if a change is suggested an uneasiness is engendered that before long modern medicine will become too great a burden for the shoulders of the older fashion of physician; the more so it is feared this will be the case if such bulky instruments as the sphygmomanometer are added to the load. If it can be shown that such instruments do really help in diagnosis, then they should be welcomed.

The purpose of this lecture is to show you that observations, instrumental in character, showing elevation of blood-pressure, are able to reveal the existence of "primary cardiac hypertrophy" when all other signs and methods fail. Without such observations twenty-four out of sixty-three cases drawn from the out-patient practice of this hospital would at the present moment be wrongly diagnosed, and be treated under the erroneous diagnosis of bronchitis and emphysema, dyspepsia, fatty heart, senile heart-disease and neurasthenia, etc. Too often such diagnoses are made rather as short cuts to the riddance from our consideration of cases that are difficult to understand, than as a result of proper estimation of the significance of the signs presented.

By occult "primary cardiac hypertrophy" is meant a condition of increased activity of the heart accompanied by an increase of the mass of its muscle, but without any of the signs hitherto accepted as proof of the existence of enlargement: this hypertrophy of the heart is accompanied by hypertension, is independent of valvular endocarditis and other well-known causes of cardiac enlargement presently to be enumerated.

Such cases are not infrequent; during the last two and a half years there have been seen by me in this department between three and four thousand new cases; a tenth of these have been proved by microscopic examination of the sputum to be tuberculous; amongst the remainder are a considerable number of cases of valvular disease of the heart, of asthma, of bronchitis and bronchiectasis, of aneurysm, and a few of malignant disease of the lungs and mediastinum; as might be expected dyspeptics and neurasthenics are not unrepresented, but in addition there have been differentiated some sixty odd cases of "primary (so called) cardiac hypertrophy," and probably more would have been found had there been time to make a more systematic examination of all the cases for elevated blood-pressure.

Signs of manifest "Primary Cardiac Hypertrophy."

The ordinarily accepted signs are the following:

(1) Alteration of the impulse of the heart. The maximum impulse of the heart is dislocated downwards and to the left; it is localised; the impulse is forcible or heaving in character.

(2) Alteration of the area of cardiac dulness. The area of cardiac dulness may be increased to the left or to the right, or in both directions.

(3) The normal cardiac sounds are altered, or there are discovered a number of adventitious sounds.

The first sound at the apex may be booming in character; the pulmonary and aortic second sounds may be more accentuated than usual and there may be a want of synchronism, so that there is said to be a reduplication of the second sound. In addition there are said to be murmurs indicating relative valvular stenosis or insufficiency, or there may be sounds which so closely mimic true murmurs as to lead only too frequently to the diagnosis of valvular defect or obstruction or leakage. These cardio-pulmonary or exocardial sounds require careful consideration, because the error they give rise to has not infrequently led to very serious disturbance of the patient's condition, in that cardiac stimulants have been prescribed with the idea that the heart was acting weakly.

(4) We are accustomed to feel the pulse in the approved fashion, and have endeavoured to dissociate that element of resistance of the pulse which is due to a thickened wall from that which is due to the force of the heart at each systole. Around this subtle investigation much controversy has been carried on, the more conservative of you who work in the Out-patient Department here feeling more secure with a digital as opposed to an instrumental investigation. What may be said with truth is this—that pulses which have been passed by you as of normal or subnormal tension by digital examination have been proved by instrumental means to have been raised far above the normal, even after making allowance of as much as 30 mm. of mercury, which is the outside limit of allowance for stiff vessels. Post-mortem examinations have proved the existence of such conditions as are accepted on all hands to be the usual and constant accompaniments of high arterial tension, viz. great cardiac hypertrophy, lengthened and toughened arteries, and renal sclerosis. To persist in the older methods of investigation in the face of such evidence is to court disaster; instead of following it would be better to abandon them; personal experience has shown me the wisdom of such action. It is quite true that a stiff vessel-wall will cause some error when the sphygmomanometer is used, but, as recent clinical investigations have shown, even with the stiffest vessels the error has not been more than 30 mm. of mercury. If you think it more accurate to make this allowance in all readings, even then you will find that there are many cases in which the pressure is higher than 120–130 mm. of mercury, and yet the finger failed to detect the condition. Personally I freely admit that I cannot correctly estimate the pressure by the finger, and that I have practically yielded to the experience gained from my own mistakes and those of others, and taken to routine use of the sphygmomanometer. If you will do this I can certainly promise you that you will be less mystified in your clinical work, because better armed with an instrument of precision, and you will be of much greater use to your patients, because you are more accurate in your diagnosis.

Assistance derived from the History of the Case.

Not only do we rely upon the physical signs to direct us to the discovery of "primary cardiac hypertrophy," but we get much help from a consideration of certain conditions which are known to lead to such change. The widespread changes due to infection may cause the heart to hypertrophy or dilate, or do both according as the infection has fastened on the valves, cardiac muscle, pericardium, or mediastinal tissues; such cases we may call cases of "secondary cardiac hypertrophy." It is right, therefore, to suspect these pathological effects when physical signs and symptoms have followed upon some acute exanthematous disease or pyæmia, septicæmia, or one of the cryptogenic infections, such as may arise by an invasion of the circulation by streptococci normally resident in the intestine. If, however, there is no history of such infection, and there is a history of polyuria occurring, especially at night, of blindness, headache, and vomiting in a patient who is a painter or is the subject of gout, then it would be right to think of "primary cardiac hypertrophy" as probably existing. Exophthalmic goitre is also able to cause hypertrophy of the heart in a certain proportion of cases, but why it does so and in such a variable proportion of the cases is a pathological problem to which it is difficult to find an answer. The remaining alleged causes of secondary hypertrophy are well known to you—alcoholism, addiction to tea, to tobacco and to athletics. But before you accept these latter very common habits as causes, it would be well for you to ask yourselves how often you have seen in the post-mortem room cases of cardiac hypertrophy which have no association with valvular endocarditis or other changes consecutive to infection, with Graves' disease, or with excess in tea, tobacco, alcohol, or athletics. In a certain proportion of the cases you will find great shrinkage of the kidney—the familiar granular kidney—and you may find the kidney to be larger than normal, but in both cases you will find fibrosis of the organ: there has been in the former a loss of the parenchyma with replacement by indifferent fibrous tissue; in the other the parenchyma has

been lost and has been replaced by fibrous tissue, but in addition repair has led also to the compensatory formation of new parenchyma—a process accepted by pathologists not only in the case of the kidney but in other organs. Returning to the subject of the so-called "athletic heart," it seems to me that, though it is commonly diagnosed, it is a very rare occurrence in the post-mortem room. I have never seen a case of cardiac hypertrophy in the post-mortem room in which the change was traceable to excessive action of the muscles alone, *i. e.* without the existence of other and more reasonable and verifiable causes.

An Impasse in Clinical Investigation.

It is quite certain that no one who follows closely the routine post-mortem examinations of a hospital will fail to observe that cases of great "primary cardiac hypertrophy" are discovered which were never suspected at the bed-side as a result of an appeal to the signs already enumerated or to the various histories above referred to. The reason is simple—*that the signs may have been obscured by accompanying emphysema, and even such readily detectable signs as tortuosity of the brachial arteries may be obscured by an excess of subcutaneous fat.*

The question then must be raised—How can this residuum of cases of occult "primary cardiac hypertrophy" be detected clinically? The answer is—By a routine and a repeated observation of the blood-pressure by means of the sphygmomanometer, hypertension being established.

Of the 63 cases already referred to *all* had raised blood-pressure; there was no constant history of such infections as scarlet fever or rheumatic fever, of exophthalmic goitre, excess in tea, alcohol or tobacco, or athletics; 39 of these gave signs which are ordinarily accepted as proof of the existence of "primary cardiac hypertrophy"; *the remaining 24 gave no evidence whatever that the heart might be larger than it should be, other than the existence of increased blood-pressure.*

It is reasonable to believe that the cause operative in the 39 cases producing clear evidence of cardiac enlargement is also present in the remaining 24 cases, although in these latter cases the cardiac enlargement cannot be made out.

The 39 cases are being treated on the lines usually adopted for such cases of primary cardiac hypertrophy, rest in recumbency being advocated with success, and being far more useful than the transitory effects of vaso-dilators. This lecture is given as a plea for treating the remaining 24 cases on the same plan.

Why and how the "primary cardiac hypertrophy" arises is not a matter for discussion on the present occasion, but when primary hypertrophy exists in a masked or unmasked condition the same mode of treatment should be carried out.

March 8th, 1909.

ON INTRA-THORACIC GROWTHS.*

By CECIL WALL, M.D.Oxon., F.R.C.P.Lond.

I HAVE recently analysed the clinical notes of seventy-six cases of intra-thoracic growth, and have arranged them in the following groups:

(1) New growths starting in the mediastinal bronchi or lungs:

(*a*) Sarcoma and lympho-sarcoma.

(*b*) Carcinoma and endothelioma.

(*c*) Hodgkin's disease.

(*d*) Mediastinal new growth in association with lymphatic leukæmia.

(2) New growths starting in the pleura.

(3) Intra-thoracic growths secondary to growths elsewhere in the body.

Sarcoma of mediastinum.—In the first group I have included all tumours of a sarcomatous nature arising from the tissues of the mediastinum or of the hilum of the lung. I would have attempted to differentiate between cases of lympho-sarcoma and those of sarcoma with oval, round or mixed nuclei had I felt certain that the pathologists, in whose debt I am, used a uniform nomenclature, or had I been able to distinguish clearly any clinical characteristics in such sub-divisions. Time and accurate observation may ultimately enable the differentiation; at present I do not feel that the data are sufficiently well established to justify the procedure.

I am not even convinced that all the cases found in this group are really of a sarcomatous nature; it seems not improbable that some of the tumours designated as lympho-sarcomatous may have had some obscure inflammatory origin. Thus, Dr. Turnbull has sent me this report upon one of the cases:

"This is not a true sarcoma. The cells show very considerable polymorphism and lie free in a reticulum which tends to fibrose. The characteristic cells of Hodgkin's disease are not present. The cells are very similar to those seen in inflamed glands but are not typical lymphocytes, *i. e.* not the typical small and large lymphocytes of the normal gland and blood.

"The tumour is either a lymphatic growth or a lymphatic inflammation like Hodgkin's disease—

* Delivered before the Medical Society of London.

the great number of plasma cells in many of the glands cut suggest that there is an inflammatory process: it is, however, an invading process, therefore if it be a growth it is a sarcoma and not an adenoma. It falls in well with Kauffmann's definition of lympho-sarcoma as opposed to small round-celled sarcoma (v. Lehrbuch, 1904, p. 145)."

In view of this report I have excluded this particular case from my series: perhaps others are wrongly included. Should this inflammatory theory be found to be justified it may lead to successful treatment of some cases at present considered incurable. In this connection I may make allusion to the astonishing success attending the treatment by the X rays of certain cases in which the diagnosis of lympho-sarcoma, or at least of Hodgkin's disease, seemed clinically justifiable.

My object to-night, however, is to discuss the clinical rather than the pathological aspect of the subject.

The cases which I have grouped together as sarcomatous or lympho-sarcomatous are thirty-four in number. In twenty-six of these the diagnosis has been confirmed by competent pathologists after microscopical examination: in the remaining eight the condition found at the post-mortem examination seemed to justify the diagnosis, though the histological examination was not complete.

Of the twenty-six certain cases twenty-three were males and three females; of the doubtful cases six males and two females.

This proportion does not bear out the statement made by Sir Douglas Powell that sarcomatous growths are commoner in females than in males.

I do not suppose that my figures represent the true proportion of sex incidence: they may be explained by difference in accommodation provided for the two sexes in the hospitals or the greater difficulty in obtaining leave for post-mortem examinations in the case of females; however, they do draw attention to the fact that it is not wise to attach much importance to the sex in differential diagnosis.

Walshe thought that both sexes were equally affected. Hare's figures show 49 males and 18 females.

The age incidences of the 26 cases was: First decade, 2; second decade, 3; third decade, 4; fourth decade, 9; fifth decade, 5; sixth decade, 3; or, if the doubtful cases are added, making 34: First decade, 2; second decade, 5; third decade, 6; fourth decade, 11; fifth decade, 6; sixth decade, 4.

The figures show that the maximum incidence was between the ages of twenty and sixty (28 out of 34). Hare's figures showed a maximum incidence between twenty and fifty (41 out of 67), but considering the smallness of the figures the difference is not remarkable. The youngest in my series was æt. 11 years, the oldest æt. 70 years.

In differential diagnosis the age of the patient may help to exclude aneurysm; apparently no age can be taken to exclude mediastinal sarcoma.

The next point which I have attempted to investigate is the duration of the disease. In twenty-two cases in which the diagnosis was certain and the clinical records gave the date of the first symptom, the total duration of the disease averaged 7·7 months. The shortest was in a boy, æt. 11 years, who had suffered from asthma for some time, but died three weeks after being forced by the dyspnœa to seek medical advice. Twelve out of the twenty-two cases lasted more than four months and less than eight from the first symptom. In two cases the symptoms persisted for fifteen months.

The duration does not seem to bear any direct relationship to the age, though the general impression from the study of the cases is that the disease tends to run a shorter course in the younger patients.

The prognosis, I presume, if the growth is sarcoma, is necessarily fatal; in some few cases where this diagnosis has been confidently made, such improvement has occurred that it has been revised. I have not yet had an opportunity of following through the subsequent history of such a case.

Symptoms.—Of the thirty-two cases in which I found records of the clinical history, in only one were pulmonary symptoms entirely absent. In this case there was a mixed-celled sarcoma of the superior mediastinum with invasion of the right lung. The mediastinal growth and the invasion of the lung had been recognised by the X rays ante-mortem, but the notes record that pulmonary symptoms were absent from beginning to end.

Cough and dyspnœa were present in thirty out of thirty-two cases. The cough is usually irritating and associated with scanty sputum; sometimes it

is paroxysmal and very distressing, occasionally it has a brassy quality.

The dyspnœa in the earlier stages is only apparent on exertion; later it may become paroxysmal or continuous and distressing. Stridor, due to pressure on the trachea and implication of the laryngeal nerves, was noted in several cases, but as a rule it is a late symptom.

On the whole dyspnœa seems to be the most characteristic pulmonary symptom of mediastinal sarcoma.

In many cases there was no sputum, or it was described as scanty and mucoid; in some instances it was copious and muco-purulent, and in one case fœtidity heralded the development of gangrene.

In 17 out of the 32 cases hæmoptysis was recorded at some period in the illness, but in every instance the note adds that the amount of blood expectorated was small and not frequently repeated.

Pain is recorded as a symptom in 15 out of the 32 cases. It is generally referred to the chest, and apparently is most frequently due to pleurisy: occasionally it is caused by implication of the thoracic wall in the growth. It is seldom so severe or agonising as the pain met with when the growth is of a nature more likely to produce metastatic deposits in the bones.

I do not think that pain can be regarded as a very important symptom of mediastinal sarcoma.

Of the pressure symptoms the most important are those resulting from venous obstruction: œdema of the face and neck in the morning which subsides during the day is mentioned several times in the records as the first symptom: marked œdema of some part of the body is recorded in 11 out of 32 cases. I believe, however, that this marked œdema, which, as a rule, only occurs late in the disease, is of less practical value in early diagnosis than that slight puffiness of the face which results from venous obstruction and leads occasionally to the belief that the patient is getting fatter.

The records show that venous distension was noticed in 14 cases: this sign is of importance in the differential diagnosis from aneurysm. It is mentioned here because of the close connection with the symptoms of œdema.

Dysphagia was found in 8 cases: this may be produced by pressure of the growth upon the œsophagus or by implication of the vagus nerve: it is difficult to distinguish the exact cause unless a full-sized œsophageal bougie has been passed into the stomach to show that there is no mechanical obstruction. I am not aware that this test was applied in any of the cases.

Signs.—With regard to the signs by which the diagnosis of mediastinal sarcoma was established in those cases, it is rather remarkable that in only 6 out of the 32 was there direct evidence of a solid mass in the superior mediastinum, *i. e.* dulness under the manubrium sterni extending either to right or left, with or without bulging in that region, and with or without tracheal breath-sounds and whispering pectoriloquy.

In 12 cases records of the radiographic examinations show that a growth was thus recognised 7 times; in 5 cases the results of the radiography were ambiguous. In 13 cases the physical signs suggested consolidation of the lung: sometimes the heart was in its natural position, and sometimes was displaced to the opposite side by a bulky mass of growth in one lung so that the signs closely simulated those of pleural effusion. In one case under my care the first signs were those indicating diminished air entry into the left bronchus. The radiogram then showed a shadow at the root of the lung: later, owing to collapse of the lung, together with implication of the left phrenic with corresponding paralysis of the left half of the diaphragm, the heart was displaced towards the affected side.

In 15 cases the examination of the chest revealed signs of pleural effusion: in 6 of these the fluid was found to be blood-stained, generally of a brownish colour, in 5 it consisted of clear serum, and in one it was purulent. In the last case the autopsy showed that the collection of pus was in the lung substance, and had resulted from softening of the growth. In the remaining 3 cases the nature of the fluid was not recorded. In 3 cases acid-fast bacilli were found in the pleural exudate when examined by Jousset's method, but no evidence of pulmonary tuberculosis was found at the autopsy. In 8 cases enlargement of the glands in the neck or axillæ was noted. Evidence of venous obstruction was noted in 8 cases only—a smaller number than might have been expected when the tendency of mediastinal growths to invade the veins is remembered.

In no case was there clinical evidence of invasion of an artery as in those recorded by Dr. Samuel West ('Trans. Path. Soc.,' vol. xxxvii, p. 143) and Dr. Hale White ('Trans. Path. Soc.,' vol. xliv, p. 14), or of cardiac valvular incompetence.

In two cases pulmonary tuberculosis complicated the condition.

In six of the cases no physical signs were found ante-mortem suggesting the presence of a mediastinal growth.

Carcinoma of mediastinum.—In the second group I have included all those cases in which the growth, after microscopic examination, has been pronounced to be carcinomatous.

Of this condition I have analysed the notes of eleven cases. In eight of these the growth is supposed to have started in or near a main bronchus; in two the primary site was regarded as doubtful, and in one the growth was supposed to have originated in the upper lobe of the lung.

Seven of the cases were in men, four in women.

The age incidence shows that all save one occurred in persons of thirty-nine years of age or more. The single exception was a case of carcinoma of the right lung and anterior mediastinum occurring in a girl, æt. 15 years, who was admitted to the London Hospital suffering from urgent dyspnœa with signs of mechanical obstruction, which had existed for three days; tracheotomy was performed but no relief resulted. The condition was too urgent to permit a detailed examination. At the autopsy a growth was found springing from the region of the right bronchus and obstructing its lumen with secondary deposits in the anterior mediastinum, the bronchial and the lower cervical glands; the pericardium was invaded. Dr. Turnbull reports that "the cells as a rule are round and suggest sarcoma, but they have not the nuclei of sarcoma cells and no intercellular substance. Further, they form chains, and after much search many secondaries have been found in which the cells are packed as in epithelioma (no prickle-cell borders) and enclose definite alveolar spaces. The cells are so like the cells lining collapsed alveoli that I think this undoubtedly a carcinoma of alveolar epithelium."

The ages of the other cases range from thirty-nine to sixty-five.

The duration was recorded in ten cases; it varied from three days, in the girl already mentioned, to ten months. Omitting this anomalous case the average duration seems to be about eight months.

Symptoms.—The prominent symptom in these cases seems to be pain, due in most instances to secondary deposits in bones or to the implication of nerve-trunks.

Excluding the case of the young girl, pain was recorded as troublesome in nine out of ten cases.

In one case there was pain in the left shoulder and wasting of the left arm, due to a metastatic deposit in a lower cervical vertebra, which had involved the brachial plexus.

In another case there was an area of anæsthesia dolorosa over the lower ribs, due to a secondary deposit in the seventh dorsal vertebra.

These are examples of the way in which pain becomes a predominant feature in cases of intra-thoracic carcinoma.

Of the pulmonary symptoms dyspnœa seems to be the most frequent, occurring in seven of the eleven cases. This is probably due to the frequency of bronchial obstruction.

Cough was troublesome in only four cases; hæmoptysis was noted in only three. In three patients no pulmonary symptoms were present; though one of these is recorded to have coughed up a stinking lump while in hospital, it is expressly stated that he had no other symptoms suggesting intra-thoracic disease.

Hoarseness was noted in two cases and in two dysphagia was present.

Signs.—The signs found on examining a patient suffering from intra-thoracic carcinoma seem to be less definite than those found in cases of sarcoma.

In four out of the eleven cases no signs of intra-thoracic disease were detected ante-mortem.

In four cases signs of consolidation of part of a lung were detected, and in two of these there was evidence of retraction of the affected portion of the lung: the retraction is to be explained by the collapse of the lung consequent upon bronchial obstruction. In three cases the signs indicated pleural effusion: in one of these when the fluid was aspirated for the second time it was found to be blood-stained.

Evidence of venous obstruction was found in one case only: in this case there were signs indicating implication of the right recurrent laryngeal and right sympathetic nerves.

The patient was a blacksmith, æt. 39 years, who

was admitted to the London Hospital in 1903, under the care of Dr. F. J. Smith. The autopsy showed a growth starting from the root of the right lung filling the superior and anterior mediastina, and invading the pericardium and the glands of the neck on the right side. Secondary deposits were found in the heart. Microscopic examination left it in doubt whether the growth was a carcinoma or an endothelioma.

There is a record of a radiographic examination in one case only, and this proved of no value in the diagnosis.

In most of the cases the secondary symptoms and signs attracted most attention. Implication of the brachial plexus, intercostal nerves, spinal cord and brain leads to symptoms which overshadow those which can be ascribed to the primary growth; examples of all these conditions are found in the series I have collected. In this connection I may allude to the tendency to metastases in bone where there is a primary carcinoma of a bronchus. Dr. Turnbull has drawn attention to this fact in his account of two cases of carcinoma of a bronchus in the last volume of the 'Archives of the London Hospital Pathological Institute.'

Hodgkin's disease.—The third group of my cases of mediastinal new growths includes those found in association with Hodgkin's disease.

It is of course well known that in Hodgkin's disease the lymphoid tissue of the mediastina may be very markedly increased and may produce the signs and symptoms of a mediastinal new growth. In my search through the records of the London and Brompton Hospitals, however, I have been able to find but two examples in which subsequent microscopical examination has established the diagnosis. A full account of one of these cases has been published recently in the 'Archives of the Pathological Institute of the London Hospital' (vol. ii, p. 141). The patient was a boy, æt. 4 years. The total duration of the illness was six months, the chief symptoms being wasting, anorexia, constipation and double otorrhœa.

The glands of the neck, axillæ and groins were enlarged and there was evidence of a new formation in the superior mediastinum which presented as a pulsating tumour at the inner end of the second and third right interspaces. There was evidence of obstruction of the superior vena cava and of narrowing of both main bronchi.

The blood-count showed 15,000 lencocytes per c.mm., of which 80 per cent. were polymorphonuclear, 11 per cent. large hyaline cells, 8 per cent. lymphocytes, and 1 per cent. eosinophile; later the leucocytosis rose to 28,000 per c.mm.

Throughout the period of observation there was intermittent pyrexia (99°–103° F.), and there was gradually increasing dyspnœa with attacks of diarrhœa and vomiting. The autopsy showed the typical changes of Hodgkin's disease, with a large mass in the mediastina which had extended between the costal cartilages without eroding them.

The second case was in a boy, æt. 16 years, who was under my care in the Poplar Hospital in 1902, and subsequently was in the London Hospital under Dr. Warner.

The total duration of the illness was twenty-one months; the first symptom was pain and swelling of the chest with signs of a mediastinal new formation. Eight months later the glands in the neck and axilla became enlarged, and one month after this the boy began to complain of dyspnœa in addition to the pain. The mediastinal tumour steadily increased in size, and pulsation became recognisable; ultimately the overlying skin became red and œdematous. Radiography confirmed the presence of the mediastinal tumour.

Towards the end there were signs indicating obstruction of the right innominate vein and of the trachea; there was in addition, during the last few days, a tendency to double adductor spasm of the laryngeal muscles.

Throughout the illness the temperature was irregular, varying from 99°–101° F. At the autopsy a mass was found in the mediastina, which had extended into the roots of the lungs, the pericardium and the chest wall.

The report of the microscopical examination describes the tumour as "lymphadenoma." I have notes of three other cases of Hodgkin's disease in which the mediastina were involved, but during life there had been no symptoms suggesting the complication.

I have already alluded to another case in which Dr. Turnbull, after careful microscopical examination, expressed doubt as to the tumour being of a sarcomatous nature or inflammatory and allied to Hodgkin's disease. This was the case of a man-servant, æt. 28 years, who had been feeling ill for

a year, and a month before his death began to complain of pain in his left arm, of paroxysmal dyspnœa and a brassy cough.

The glands of the left side of the neck and left axilla were enlarged; there were signs of compression of the left axillary vein with œdema of the left arm.

In the chest there were signs of consolidation of the apex of the left lung and of a tumour in the superior mediastinum; radiography confirmed these signs. Later, signs of pleural effusion developed. There was slight pyrexia (98·4°–100° F.); the blood showed no leucocytosis. At the autopsy there was found a large mass occupying the anterior and superior mediastina; the left bronchus was obstructed and the left lung collapsed; there was a large left pleural effusion.

The growth had not invaded the lung substance. The glands on the left side of the neck and in the left axilla were enlarged.

I have already given Dr. Turnbull's reasons for hesitation in classifying the growth.

Lymphatic leukæmia.—The fourth group of cases includes three examples of a condition to which allusion is usually made in articles dealing with growths of the mediastinum, but of which I have not been able to find any examples in the literature available to my hands.

I may be pardoned, therefore, if I briefly recount the cases.

The first was in a girl, æt. 16 years, who was admitted to the London Hospital in 1903 under the care of Sir Stephen Mackenzie. The total duration of the illness was fifteen weeks; the first complaint was of swelling of the right side of the neck and of a lump in the left axilla. There was a slight cough with scanty brown sputum. A month after the onset she began to complain of dyspnœa.

On admission enlarged glands were found in the posterior triangles of both sides of the neck, and there was evidence of a tumour in the mediastinum with bulging of the manubrium sterni; there was evidence of obstruction of the left innominate vein. The spleen was palpable.

The temperature at first ranged from 99°–102°F.; later it was normal.

The blood-count showed 1,300,000 erythrocytes and 100,000 leucocytes to the c.mm. The white cells are described as being practically all lymphocytes.

At the autopsy, in addition to the enlargement of the cervical axillary and mesenteric glands, there was found a growth in front of the trachea surrounding the vessels of the superior mediastinum and invading the pericardium.

Microscopically the growth was described as consisting of small round cells.

The second case was that of a man, æt. 20 years, by occupation a clerk. He was admitted to the London Hospital in 1907 under the care of Dr. Hutchison complaining of swelling of the neck.

The total duration of the illness was one month.

A week before his death purpura developed and two days later the gums began to bleed. Dyspnœa and cyanosis also developed.

The glands of the neck, groins and axillæ were enlarged, and there was evidence of a growth in the mediastinum obstructing the superior vena cava and the trachea.

The temperature ranged between 99° and 100°F. The blood examination showed 5,750,000 erythrocytes and 580,000 leucocytes per c.mm., with 65 per cent of hæmoglobin. The white cells were almost entirely large lymphocytes. At the autopsy large cervical axillary and cœliac glands were found, and a large mass in the anterior and superior mediastina infiltrating the pericardium. Dr. Turnbull reports that the microscope revealed the changes usually met with in acute leukæmia.

The third case is of a somewhat different nature. A boy, æt. 11 years, was admitted to the London Hospital in 1908 under the care of Dr. Dawson. The total duration of the illness was eight weeks, and the earliest symptoms pains in the knees and swelling of the neck.

There was paroxysmal nocturnal dyspnœa with much stridor; purpura of the arms and legs developed and also œdema of the left side of the chest.

The glands on both sides of the neck were enlarged and also those in the axillæ and groins; there was evidence of a new growth in the superior mediastinum with obstruction of the superior vena cava.

The temperature was irregular, varying between 98°–101°F. The blood examination showed 70 per cent. lymphocytes, but unfortunately was not very satisfactory.

At the autopsy there was found a malignant growth of the marrow-cells eating through the

lower end of the femur, which Dr. Turnbull considers to be a malignant myeloma.

All the glands in the body were enlarged, and there was a large mass occupying all the mediastina and invading the pericardium.

Dr. Turnbull reports that unfortunately he had not got a satisfactory blood-film to examine, but such as it was it showed 51·3 per cent. large lymphocytes, 34·6 per. cent. small lymphocytes, 3·88 per cent. myelocytes, 10·1 per cent. polymorphonuclear leucocytes. The marrow of the femur had broken through the shaft and invaded the periosteum, and the tissue round the glands and the walls of the gut, etc., were invaded by cells resembling myelocytes.

New growths of pleura.—The next group includes five cases in which the diagnosis of primary malignant disease of the pleura was made at the post-mortem examination. Unfortunately, in only two of the cases was the result of the microscopical examination of the tumour recorded.

Of the five cases four were male. The ages were 40, 51, 65, 67 and 73. The duration of symptoms ranged from two months to six months, the average being about four months. In all the cases the pulmonary symptoms and signs suggested pleurisy with effusion. In one the effusion was at first clear, in three it was blood-stained, and in the fifth the nature of the fluid was not recorded.

Symptoms and signs due to secondary deposits seem frequent, as in the cases of primary cancer arising in the mediastinum.

Thus in a man, æt. 67 years, who suffered from an endothelioma of the right pleura with a large blood-stained pleural effusion, deposits were found in the glands of the right axilla and in the right pectoralis major which were recognised before death.

In another man, æt. 40 years, who at first had signs of a large left pleural effusion and ultimately developed a pyo-pneumothorax, there was a primary endothelioma of the left pleura with metastatic deposits in the brain, kidney, and pancreas. The secondary growth in the brain produced the signs of cerebral tumour with epileptiform convulsions.

In the third case of a man, æt. 51 years, a large blood-stained pleural effusion was associated with multiple subcutaneous nodules, which were found to show the structure of an alveolar carcinoma. The post-mortem diagnosis was primary malignant disease of the right pleura with secondaries in the left lung, spleen, kidneys, supra-renals, mesentery, pericardium, and subcutaneous tissues.

In the case of another man, æt. 65 years, suffering from a primary malignant growth of the right pleura invading the ribs, subcutaneous nodules were found before death, and subsequently secondaries were found in the lungs, left kidney, right supra-renal, liver, and stomach. The nature of the growth is not recorded.

I regret that in none of these cases was there a record of a cytological examination of the pleural exudate. In one case that I have seen, but for other reasons was not able to include in this series, definite cancer-cells were found in the fluid aspirated from the pleural cavity, and, of course, in the literature of the subject several examples may be found where the examination was of use in the diagnosis.

Secondary intra-thoracic growths.—I have notes of eighteen cases in which secondary growths were present in the lungs, the primary growth being elsewhere.

The number is insufficient to permit any important clinical deductions: a few observations may be of interest. In six cases there had been a primary sarcomatous growth in a long bone, and in five amputation had been performed. Five of the six patients were female. The ages varied from fifteen to thirty-five, but five of them were under twenty-two years of age. In one case the first pulmonary symptoms developed eight months after the primary growth was first noticed, in two there was an interval of ten months, in two of eighteen months, and in one of two years.

In five out of the six cases cough, hæmoptysis, and dyspnœa were the only pulmonary symptoms: in two cases a blood-stained pleural effusion was found: in the four other cases the signs indicated masses of consolidation in the lungs. In one the growth was so large that the heart was displaced to the opposite side, and the signs very strongly suggested pleural effusion. No fluid was found on exploration, and the autopsy showed that none was present. In another case while under observation the growth, of which the diagnosis had been confirmed by the X rays, broke down and led to the production of a pneumothorax.

Of the remaining examples of secondary sarcoma, in two the primary growth was presumably in a

rib: both patients were men aged fifty-three and sixty respectively. In one there was a serous pleural effusion, and there was a tumour connected with the fifth rib with secondaries in the glands of the axilla and posterior triangle on the same side. Before the development of the effusion pleuritic friction was heard on auscultation over the costal tumour.

Post-mortem, nodules of growth were found in both pleuræ scattered through both lungs and both kidneys. Unfortunately no microscopical examination was made.

In the second, a man, æt. 60 years, the first sign was a mass found in connection wtth one of the ribs: later masses developed in the clavicle and in the right breast, and there were signs of massive consolidation of the underlying lung. Post-mortem examination was refused.

In another case a man, æt. 38 years, noticed first a swelling of the right clavicle: he died ten months later with symptoms suggesting a secondary deposit pressing on the brain. In the interval he had developed swellings on the opposite clavicle, the parietal bone, the sternum and the cervical glands.

There were no pulmonary symptoms, but at the autopsy in addition to the above growths it was found that there was a large deposit in the mediastinal glands, with compression of the bronchi and invasion of the lump from the root. Deposits containing calcareous spicules were found in both lungs. The growth was described as a periosteal sarcoma, but I do not know whether a microscopical examination was made.

In a woman, æt. 47 years, who was admitted to the hospital complaining of pains in the chest and had symptoms of heart failure, there were found signs of consolidation and retraction of the apex of the left lung with a large and nodular liver. Shortly after admission she developed blindness and proptosis of the right eye. She lived about four months from the first development of symptoms. At the autopsy there was found a primary spindle-celled alveolar sarcoma of the dura mater of the right middle fossa, with extensive secondary deposits in the glands at the root of the left lung. The left lung was largely replaced by growth; there were secondary nodules in the pleuræ and in the liver.

In the remaining examples of secondary sarcoma one followed a sarcoma of the back which had been removed four months before; there was a large blood-stained pleural effusion, and post-mortem extensive deposits were found in the lungs, pleuræ and mediastina. The notes are not complete, and it is not quite clear that the growth in the back was really the primary one.

Another case was in a girl, æt. 18 years, who had a primary cystic sarcoma of the kidney, but though there were no signs or symptoms suggesting pulmonary disease there were numerous deposits scattered through both lungs.

The last case is that of a man who had a primary growth in the left kidney, and for nine months had pulmonary symptoms—cough, hæmoptysis and dyspnœa. He developed a large blood-stained pleural effusion which was tapped several times. There were numerous secondary deposits in both lungs.

I have collected five examples of secondary cancer of the lung and mediastinum.

The first is of interest from the point of view of diagnosis: A man, æt. 53 years, was admitted to the London Hospital under my care with symptoms pointing to œsophageal obstruction; there was evidence of a new formation in the mediastinum and the left vocal cord was paralysed. There was dulness and deficiency of air entry into the left lower lobe; the aortic second sound was accentuated. The radiograph seemed to show that there was an aneurysm of the third part of the arch of the aorta. On post-mortem examination there was found a primary epithelioma of the œsophagus with extensive secondary deposits in the mediastinal glands, which had ulcerated into the bronchus and had caused retention of secretion in the left lower lobe.

The second case was one of secondary carcinoma of the anterior mediastinum compressing the pulmonary artery and the superior vena cava. The primary growth was in the breast, which had been removed two and a half years before the development of pulmonary symptoms. These symptoms were cough, paroxysmal dyspnœa and hæmoptysis; towards the end, eight months later, a pleural effusion developed.

In the third case a man, æt. 19 years, five months after removal of the testis for carcinoma, developed signs of thrombosis of the inferior vena cava, which was found to be due to secondary deposits into the abdominal glands which had invaded it.

There were many secondaries in various parts of the body, and large deposits in both lungs, but during life there had been no pulmonary symptoms.

The remaining two cases are examples of pulmonary growths secondary to carcinoma of the rectum—one in a man of thirty, who also had a secondary deposit in the cerebellum. The cerebellar symptoms were predominant, but there were signs of consolidation of part of the right lung, and at the autopsy there were found many deposits in the lungs, pleuræ and bronchial glands, in addition to numerous deposits elsewhere in the body.

The last case was in a man, æt. 52 years, who, thirteen months after colotomy for carcinoma of the rectum, developed a large blood-stained pleural effusion, and the signs indicating intra-

thoracic secondary deposits. In this case leave for a post-mortem examination could not be obtained.

A study of these cases of secondary intra-thoracic growths serves to show what, of course, is well known, that extensive growths may exist in the lungs without either symptoms or signs. It also shows that the deposits are more often in the lungs than in the mediastinum, and that the pleura is not uncommonly affected. It emphasises the importance which we should attach to such symptoms as dyspnœa or hæmoptysis, and such signs as those of pleural effusion in patients who are known to have suffered from sarcoma or cancer in another part of the body. Sarcoma of the mediastinum is a disease chiefly of the middle period of life. It is generally associated with pulmonary symptoms, of which cough and dyspnœa are the chief and hæmoptysis not uncommon. Symptoms due to the bulk of the growth are often present, such as stridor, dysphagia and œdema from venous obstruction. Pain which can be ascribed to metastatic deposits is seldom a prominent symptom. The signs usually indicate either a pleural effusion, which is not uncommonly brown from altered blood-pigment, or the presence of a solid mass in the mediastinum or lung. Evidence of impairment of function of various structures of the mediastinum is frequently found. Cancer of the mediastinum occurs somewhat later in life. Pulmonary symptoms are less commonly prominent, and metastatic deposits in bones or implicating nerves usually bring the patient under the notice of the physician. Signs of pulmonary or mediastinal disease are often absent; if present they tend to indicate obstruction of a bronchus or an effusion resulting from pleural metastases rather than massive deposits in the lung or mediastina. In Hodgkin's disease and acute lymphatic leukæmia the presence of a mass of lymphatic gland-tissue in the thoracic cavity is merely accidental, but when present might produce symptoms and signs owing to its bulk. Cancer of the pleura seems to be a disease of later life tending to produce symptoms, as in the case of mediastinal cancer, from metastatic deposits and signs of pleural effusion. The series of cases of secondary growths in the lungs is sufficient to show the great variety of symptoms and signs that might be present, but too short to permit any general deductions.

Mr. CHARTERS J. SYMONDS asked whether Dr. Wall had found, as a matter of diagnosis, that the recurrent laryngeal nerve was more subject to pressure, as recognised by laryngeal paralysis, in mediastinal growth than in aneurysm? He was interested in that subject from the paralysis found in œsophageal disease, and occasionally where there was cancer of the œsophagus without obstruction it was a matter of importance to have the question of frequency before one's mind. Another question in which he felt interested was, Could one recognise the difference between aneurysm and growth by the X rays? He hoped so. With regard to dysphagia and the passage of bougies, it had fallen to his lot to only once pass a bougie in aneurysm, and the sensation communicated to his hand was that of passing over a spherical body. He asked whether Dr. Wall had found any value in tracheotomy for the relief of the dyspnœa in those cases? He had been disappointed in its value for aneurysm, and it had long been his intention to try a suggestion which had been made by Professor Gluck, of Berlin, who had a case of complete stenosis of the trachea in a child. He excised a rib over the lung, made an adhesion between the lung and the surface, and then opened the lung and created a large bronchial fistula, through which the child breathed comfortably and well. Perhaps Dr. Wall's patients were too ill for such a procedure, but it was such an interesting suggestion that he was looking out for a case in which he might try it, and it was possible it might relieve some of those cases.

Dr. F. PARKES WEBER said that Dr. Wall's remarks about lymphadenoma (Hodgkin's disease) occurring in the form of a mediastinal tumour interested him particularly. But when he summed up at the end, Dr. Wall implied that when lymphadenoma or Hodgkin's disease formed a mediastinal tumour, it was merely that one part of the disease chanced to be in the mediastinum and to form a tumour there. He did not quite agree with that. No doubt that was occasionally the case, but he believed that primary mediastinal tumours sometimes showed the histological characteristics of lymphadenoma or Hodgkin's disease. It would take too long to go into that question in detail, but he pointed out that cases which were supposed from the clinical point of view to be typical examples of Hodgkin's disease or lymphadenoma had during the last seven years been carefully examined by modern microscopical methods in different parts of the world, especially in America and England, and a characteristic histology had been discovered in them. Their main histological characters had been described by Andrewes, Reed, Longcope, etc., and more recently by Kidd and Turnbull, at the London Hospital. And these characters must, he thought, be accepted now as the true criterion for the disease. Last year he described the case of a young woman, æt. 21 years, who had a mediastinal tumour, and whom he showed as an example of mediastinal tumour with swelling of the extremities at the Royal Society of Medicine (Clinical Section, March 10th, 1908). At that time there were no enlarged lymphatic glands noticed; in fact there was no other tumour anywhere in the body, except the mediastinal one. After some time she died (in October, 1908), the final cause of death

having been acute pericardial serous effusion. The mediastinal tumour at that time weighed 66 ozs., and was not merely a conglomerate mass of glands, it was a dense tumour with a nodular surface. It was true that before death there were one or two glands to be felt at the base of the neck and in the axilla. One of them removed for diagnostic purposes as well as the mediastinal growth itself removed after death corresponded to the description of lymphadenoma (Hodgkin's disease) as given by the authorities he had mentioned. Nevertheless the disease began as a tumour in the mediastinum, and the enlarged glands formed merely a minor and later feature in the case. He exhibited coloured drawings of the microscopic specimens, prepared by Dr. J. C. G. Ledingham from the case, which showed especially the dense crowding of the mediastinal tumour and cervical glands with eosinophile cells. One of the pictures represented the bone-marrow, which did not contain any growth, but proved the existence of a decided eosinophile reaction. In a paper on acute cases of Hodgkin's disease, in the last volume of 'St. Bartholomew's Hospital Reports' (vol. xliii, p. 81), he had pointed out that a great character of Hodgkin's disease was that it was not a generalised disease of all the lymphatic glands throughout the body at first, but that for the clinical diagnosis of Hodgkin's disease there must at first be some regional localisation of the main disease, *i.e.* the lymphatic glandular tissue in some one part of the body must be particularly enlarged so as to constitute a local tumour-formation. Cases in which the lymphatic glands all over the body were slightly enlarged, without any local tumour-like growth, were not, he believed, examples of lymphadenoma, but of some other disease, possibly, for instance, early stages of leukæmia. He was surprised that Dr. Wall did not mention among the symptoms of mediastinal tumours the condition of so-called pulmonary hypertrophic osteo-arthropathy, which constituted a very striking feature in the case of his own just referred to; in that case the diaphyses of some of the bones in the hands and feet were almost doubled in thickness (as shown by the X-rays) owing to new bone-formation in the periosteum. (Dr. Wall said osteo-arthropathy was present in one of his cases.) Another curious feature of the case he had referred to was the presence of a milk-like effusion in the left pleural cavity, but at the sixth tapping the milky appearance was no longer present, and the effusion from that time was clear until the patient's death. The "milkiness" at the first five tappings was due to the presence of exceedingly fine fatty particles (very much more minute than those present in true chylous effusions), and it could be removed by shaking up the fluid with caustic soda and ether.

Dr. CECIL WALL, in reply, said the question of recurrent nerve paralysis was one to which he had devoted considerable attention in working out the statistics, and he started off with the idea that recurrent nerve paralysis practically invariably indicated aneurysm and almost excluded mediastinal new growth. He had been considerably surprised to find recurrent nerve paralysis in both sarcoma and cancer. In the former condition three or four had involvement of the recurrent laryngeal, and in the cancer cases two had that paralysis. The X rays had proved very valuable in the past in the diagnosis of new growth from aneurysm, and he believed in the future their help would be even greater, on account of the development of the use of the screen.

That not only showed whether there was pulsation, but also the border of the shadow. But the use of the rays and the interpretation of the picture required special knowledge, and he liked to compare the impressions of the expert radiographer with his own. In one case tracheotomy was performed, namely in the case of carcinoma in a young person admitted with very urgent dyspnœa, and the operation failed to give any relief. The question of the mediastinal mass in Hodgkin's disease being accidental turned on the pathological question, which he had purposely avoided in his paper, which was intended to deal with the clinical aspect.

By the use of the term "accidental" he meant that Hodgkin's disease might start in any mass of glands in the body. The second case of Hodgkin's disease to which he referred was one in which for two years the only signs found were those of a mediastinal mass, and it was not until within a few months before death that the glands in the axillæ and groins were felt. That case he had the privilege of watching during the whole of the two years. In the early stages the simulation of the case to one of aneurysm in the first part of the arch of the aorta was so close that they were sometimes in very great doubt whether it was not aneurysm, notwithstanding that such a condition would be very unusual in a boy aged sixteen.

He had seen one case of hypertrophic pulmonary osteo-arthropathy in association with new growth in the thorax. The other cases of hypertrophic osteo-arthropathy he had seen were not associated with new growths in the lung. He had been very much struck in looking over the records, especially those of Brompton Hospital, where clubbing of the fingers was always looked for and recorded if present, with the fact that it was seldom present in association with mediastinal tumours. In the 76 cases there was not one in which clubbing was recorded. He did not meet with a case in which the effusion was declared to be milky; in most of them the fluid was blood-stained, or brown from the presence of blood-pigment.

March 8th, 1909.

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A CLINICAL LECTURE ON PERNICIOUS ANÆMIA.

Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P., Physician to the Hospital.

GENTLEMEN,—I want to talk to you this morning about a disease of which we have had a good many cases in the last year or two. The first I shall mention is that of a man named Allen H—, æt. 39 years, who was admitted under us on September 27th, 1907. The story was that he was an old soldier, who had been in India and South Africa, but had never had either malaria or syphilis. He had been in perfectly good health up to two months before we saw him. He said that on a particular day, August 5th, he had first noticed weakness, and from that date he became rapidly worse; his breath had become short, and for the last month he had been in Woolwich Hospital, from which he was transferred here. When he came to us the feature which we noticed especially was his extreme pallor, combined with a lemon-coloured tint all over his body. He was well nourished, in fact he was quite plump, and apart from the dilatation of the heart, which was marked, and retinal hæmorrhages, which were easily seen, there was no sign of organic disease. Therefore the features in his case were extreme anæmia, preservation of the body nourishment, that is to say of the fat—in other words, absence of wasting and dilatation of the heart and retinal hæmorrhages. Of course in such a case as that you examine the blood nowadays, and we found in his blood the characteristic changes of pernicious anæmia. That I shall not go into at this minute. At first the red count was only 500,000 instead of 5,000,000 per cubic millimètre, but it rapidly improved, so that in a month it was 1,500,000, and by the end of November it was

3,100,000. At that date he was discharged from here to the Swanley Convalescent Home, when he said he felt well. And he went on well, he said, although always pale. He was able to go about and do his work until about the middle of October, 1908, that is to say a little more than a year from the time when he first came under us, when he again began to feel weakness. Three weeks after this his doctor told him to come back to us. He did so and was admitted again on November 6th. When he was re-admitted his condition was not so bad as it had been on the occasion of his first admission, for he had 880,000 red cells as against 500,000 on the first occasion. Otherwise he was in much the same condition as before; he still had retinal hæmorrhages. At first, in this his second visit, he improved as he did before. On November 17th, that is to say ten days after his admission, he had 1,000,000 reds, a decided increase, but from that time he began to lose ground, his reds went down, and at last, on December 4th, he had only 364,000 reds. On December 8th he died.

Very soon after that man went out in 1907 another man came into the hospital named Samson R—, æt. 52 years, a gunsmith by occupation. He was a very respectable man and had been in his present position for a long time. He was apparently quite well until six months before admission, when he began to have morning sickness. That went on for a little while; there was no hæmatemesis with it. After that he began to get short of breath, and for that he came to us. He was spotted by the house-physician and sent into the ward. He looked a little pale, but not very much so. And I would remind you that Professor Osler describes some cases of pernicious anæmia which are not pale, but rather look red, and I suppose that is due to dilatation of the capillaries, and not due to the presence of a large amount of hæmoglobin in the blood, it is probably the facies similar to that of the ordinary bus-driver. We found no other organic disease about this patient. He was in practically the same condition as the last man, he had a similar greenish tint, and was a big man and well nourished. We looked at his blood and found the changes of pernicious anæmia. His red cell count was not nearly so bad as that of the last patient; it was 1,708,000 on his admission. He rapidly improved, and on January 25th he had over 4,000,000, and in February 4,900,000 red cells, with a hæmoglobin index which was above the normal. He was discharged, and he remained well until December 8th, 1908, when he came back to us with the same story, that he had been well able to do his work until three weeks ago, when he again began to feel weakness, and to get pale. When he came back he was found to have a red cell count of 1,260,000 to the cubic millimètre, so you see it was not quite so good as when he came in the first time. We hoped that as he had improved so rapidly before he would do so again. He was put to bed, in the same bed that he had previously occupied, and he was treated in the same way. But in spite of that he did not improve, but went downhill steadily from the time he came in, and died on January 3rd, nearly a month after admission.

The third case we had was that of a woman who was admitted in July last year. Her history was rather a different one. She said she had been perfectly well until December, 1907, when she began to feel a certain amount of numbness about her limbs and staggered in her walk. In January she noticed, as other people also noticed, that she began to get pale, and at the same time her catamenia ceased, and she began to have very obstinate and repeated vomiting, not simply vomit after taking food, but always. And that went on until she was admitted into this hospital in July. She looked quite different from the other cases I have mentioned, except that she had extreme anæmia and that slight lemon tint. But she was wasted almost to a shadow, and when she first came in I was very doubtful about the diagnosis. I thought of cancer at first, for not only had she repeated vomiting and this extreme wasting, but she also suffered great pain in the abdomen, and the abdomen was extremely tender to the touch. However, on examination under chloroform nothing was found which would further the diagnosis of cancer. And then I thought also of Addison's disease, which you know begins with extreme muscular weakness and with vomiting. And that diagnosis was a little favoured by the fact that she had on her abdomen a large number of brown patches like freckles, in fact her abdomen was freckled all over, but she had no regular and general bronzing, no black spots on the mucous membrane, such as are to be seen in an ordinary case of Addison's

disease, and the matter was settled by the blood count. In the blood we found the change characteristic of pernicious anæmia, which you do not find in Addison's disease. She was extremely ill when she came in, and she died eighteen days later. After death there was found, as is usual in these cases where the gastric symptoms are marked, nothing to account for them; the stomach and intestines were, to all appearances, thoroughly healthy.

Those three cases are typical of what we call pernicious anæmia. In regard to what constitutes the disease I thought I could not do better than read you the description which was written fifty-three years ago by Addison, who was the English physician at Guy's Hospital, who first described it: "It makes its approach in so slow and insidious a manner that the patient can hardly fix the date of his earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted. The pulse perhaps large but remarkably soft and compressible, and occasionally a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness on attempting it. The heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth and waxy appearance. The lips, gums and tongue seem bloodless. The flabbiness of the solids increases, the eyesight fails, there is extreme languor, faintness supervenes, breathlessness and palpitations being produced by the most trivial exertions and emotions. Some slight œdema is probably perceived about the ankles. The debility becomes extreme, the patient can no longer rise from the bed, the mind occasionally wanders, he falls into a sort of half torpid state, and at length expires. Nevertheless to the very last, and after a sickness of perhaps several months' duration, the bulkiness of the general frame and the obesity often present the most striking contrast to the failure and exhaustion observable in every other respect." The symptoms which Addison does not remark upon—and one or two of them were observable in these cases—are, in the first place, the frequency of intestinal and gastric complications. In that one case of mine, that of the woman, there was great pain and repeated and obstinate vomiting; nothing stopped it. In many other cases there have been repeated attacks of diarrhœa, so much so that intestinal poisoning has been thought to be at the bottom of the disease. Another symptom, which was very marked in the first case which I have narrated to you, is pigmentation. The man, Allen H—, whose case I detailed to you first, had a most remarkable black and white scrotum; part of it was very dark indeed, almost like a negro's scrotum, and the other parts were leukodermic, so that his scrotum was really black and white. That was the only part of his body which was pigmented. The third symptom which is very frequent is some sort of nervous disorder. In the first man the chief point was his apathy. He had slowness of speech and great somnolence, so that he could only with difficulty be roused. In the woman the condition began with numbness and staggering. Her reflexes were natural, and although there was a certain amount of paræsthesia there was nothing very objective, and she was too ill for us to see how she could walk when she came into the ward. In other cases I have seen evidences of spastic paraplegia, with increased reflexes and dragging of the feet along the ground. In other cases people have described—though I have never seen such a case—symptoms like those of tabes. Those are, of course, spinal symptoms. So this disease not only frequently presents the general cerebral torpor and apathy with delirium which might be accounted for by the extreme weakness, but it also presents certain spinal symptoms, which apparently depend, and have been proved in some cases to depend, on a certain amount of sclerosis of the cord. In such cases, as I have told you, we now always at once examine the blood. In Addison's time that was not known, nor even in Biermer's time in 1872, when he drew the attention of the profession afresh to this great disease, and produced an admirable paper on the subject. Even then the exact changes in the blood were still unknown. But Ehrlich, who has taught us so much, has taught us how to recognise this disease among other things. We look at the red cells and find them extremely decreased in number. The red cells in one of these patients went down to 364,000 per cubic millimètre, instead of the normal five millions. At the same time the cells are altered in size and in shape—the condition which we call poikilocytosis. Many of them are larger than normal, many are

smaller and irregular in contour. That happens also in chlorosis, though not to the same extent as in pernicious anæmia, and the cells in chlorosis are not so large. And many of the red cells are nucleated. They may be of three different varieties: either the natural size, what we call normoblasts, or they may be smaller (microblasts), or they may be—and this is a characteristic of the disease—larger than natural (megaloblasts). Those will all occur in the same case. Along with that the hæmoglobin is greatly decreased, but not in the same proportion as the cells. That is the great distinction between this disease and chlorosis: that in chlorosis the hæmoglobin is decreased much more than the red cells, in pernicious anæmia the red cells are more decreased than the hæmoglobin. In consequence what we call the colour index, or the worth of the corpuscles, is usually above the normal. In the white cells there is not the same amount of change. Usually the white cells are not increased in number. In a differential count you will often find that the lymphocytes are out of proportion to the other cells. For instance, in the second case which I told you of, that of Samson R—, the lymphocytes were very much increased, that is to say, at different countings, 44 per cent., 30 per cent., 35 per cent., 59 per cent., 51 per cent., 38 per cent., which is a good deal more than natural. That is the characteristic change. The same thing happened in the case of the woman, who had 46 per cent. of lymphocytes. So two out of our three cases showed that symptom.

A third peculiarity was found in the case of Samson R—, a peculiarity which at one time led to some doubt about the diagnosis. That was the remarkable increase in eosinophile cells. They are noted in his case as sometimes being about the natural, 3 per cent., 5 per cent., 8 per cent., 6 per cent., but on two occasions they were found to be as much as 13 per cent. and 23 per cent. of the total leucocytes. That is very unusual. I have never seen a case of that kind in pernicious anæmia. The reason why that led to doubt as to the diagnosis is this: first there are forms of severe and fatal anæmia which we know to be produced by intestinal parasites. Two, at least, are well known. One is what is called ankylostomiasis, and is produced by the *Ankylostomum duodenale*. It lives high up in the small intestine, and it is very common in Egypt and is also found in many parts of Europe, especially where people are mining. Of recent years it has been found in the tin mines of Cornwall. That produces very severe anæmia of the chlorotic type, that being the type in which the red cells are decreased and the hæmoglobin decreased in much greater proportion. That is one form of anæmia due to intestinal parasites. The second is that due to the *Bothriocephalus latus*. It is a kind of tape-worm, and is found about the Swiss lakes and the Baltic Sea, and has even been found in Ireland, but never, I believe, in England. That produces a form of anæmia which is microscopically indistinguishable from pernicious anæmia, and which kills people. The condition of the blood is the same as that found in ordinary idiopathic pernicious anæmia of unknown origin. The second point is that we have recently found out that tape-worms produce eosinophilia; they increase the number of eosinophile corpuscles in the blood. That is a piece of knowledge which has been acquired in the last few years. And therefore, when we found a case of pernicious anæmia with eosinophilia of course a doubt arose as to whether it was not due to some intestinal parasite. And with that idea we gave vermifuges, and examined the stools microscopically for ova, but without result. His case remains a singular exception to the general rule that in pernicious anæmia the eosinophile corpuscles are not increased.

There is a third symptom of this disease which two, at any rate, of these cases illustrate, and that is the feature of recurrence. Two of these persons had become almost well and remained well for a year, and then came back and died. And that happens repeatedly. I have seen it in many cases now, both at this hospital and in private practice. And you will find that although the first time you see the patient you obtain great prestige from having cured him of a very violent illness, yet if you have the bad luck to see him when he again requires treatment—that is to say, if he does not go to somebody else—that prestige runs the risk of being lost, for exactly the same treatment pursued in all these cases when they returned was followed by a totally different result. When they first came they appeared to be cured by the treatment, yet when they returned it seemed to have no effect on the illness and they died.

I will now give you what musicians call a few variations on the theme; I will tell you about one or two odd cases which we have also had recently. One of them was a young woman, æt. 25 years, named Frances D—. She exemplifies a causation which is well known to occur in certain cases, and that is that pernicious anæmia, or something very like it, occurs after confinement. She had been confined at Christmas and the child was healthy. The woman had been pregnant once before and that child was healthy also. Ever since the birth of the second child she had had a vaginal discharge. For the first three weeks she had had diarrhœa, and on March 24th she had œdema of the legs, which œdema subsequently began to spread to the arms. When she came to us on April 7th, 1905, she was looking very ill, and she was obviously extremely anæmic. She, like the others, had retinal hæmorrhages, and a purulent vaginal discharge, with a sloughing sore on the perinæum. Instead of her having, as one would expect, a septic condition of the blood, with a great increase of the polymorphonuclear leucocytes and fever, she had the characteristic blood of pernicious anæmia, with a leucocyte count of 5000, and a red cell count of 700,000 only, with a high colour index, 1·4. From that condition she rapidly recovered, the ophthalmic hæmorrhages cleared up, and she was discharged on May 16th, only six weeks after admission, apparently in perfect health. She is in perfect health now, nearly four years later. But I should say that her blood never contained any megalocytes. That is the first variety I wanted to give you, that which is consequent upon confinement.

The second case is this: In September, 1908, we had a young fellow, æt. 25 years, George A—, in for deep jaundice, with bile in his urine and the usual symptoms of obstruction to the bile-ducts. The diagnosis of the cause of his jaundice was not clear, but that is true of most cases of ordinary catarrhal jaundice. There is no symptom which makes you feel certain of the diagnosis. On October 5th, shortly before his discharge, his blood was counted, and he was found to have 3,800,000 red cells, 10,800 white cells, and hæmoglobin 89·1 per cent., and, therefore, a colour index above the normal, that is to say 1·3. That was the only symptom which appeared suspicious when he went from us to our Convalescent Home at Swanley. Soon after his reception there, on November 2nd, he began to have oozing from his gums, his gums were bleeding and spongy, and at the same time our medical officer there, Dr. Smith, of Farningham, noticed that he had a systolic murmur at the base, that is to say an anæmic murmur. The bleeding went on, other symptoms appeared, and he was accordingly sent back to us at the hospital on November 10th. When he arrived back here he was of the colour of an ordinary pernicious anæmia case, and his temperature was a little raised. His pulse was rapid, and the heart dilated. He had not only bleeding from the gums, but a purpuric rash on the legs, and to some extent on the abdomen. There was no enlargement of the spleen or other sign of organic disease. He became worse and worse until he died, his death taking place on November 24th, with all the symptoms of pernicious anæmia. He had on November 17th 660,000 reds, 3600 whites, a colour index of 2, and nucleated red cells. One can hardly say that he had pernicious anæmia in the hospital in October, but it appeared to have come on rapidly, and he seems to have failed almost within six weeks.

The last case I shall quote to you in this lecture is that of a man who was admitted with violent hæmatemesis. He was a butcher. He said he had been feeling a little languid for a week or two. He drove to the market in Smithfield to do his marketing, and was then seized with hæmatemesis to a large amount, and was brought into the hospital. He had had no symptoms whatever of gastric disease, and I took it, from the appearance of the man and his age, that it was probably a case of cirrhotic hæmatemesis. But we made a count of his blood, and we found his blood was to some extent like that of pernicious anæmia. He had 925,000 red cells, 20 per cent. of hæmoglobin, and therefore a colour index greater than 1. He had 58,600 leucocytes, and in counting 200 of these there were found 5 megaloblasts and 26 myelocytes. That man became worse and worse and died. And by the time he died I had begun to believe that he was a case of fatal hæmatemesis from pernicious anæmia—a very unusual event. When we came to do the post-mortem examination we found an eroded vessel lying at the bottom of a very narrow ulcer which had been entirely latent during life. I think that in all probability the changes in the

blood were due to the great loss of blood from the gastric ulcer.

Now a few words as to treatment. These cases I practically always treat in the same sort of way, namely, by arsenic, of which I give increasing doses up to ℥x of the liquor arsenicalis three times a day. But in addition I give them red bone-marrow. It is taken out of the cancellous texture of bones and given in specially prepared capsules. In addition to that I give raw meat-juice, which can be easily mixed up with the patients' food so that they will not notice any unpleasantness from it. I also generally give them red wine, if they can take it. Those are the methods of treatment which cure these patients, and they are the methods of treatment which, when the patient comes a second time, fail. The treatment is exactly the same, as I have already told you, in cases which recover and in the cases which die. I suppose one is justified in laying the flattering unction to one's soul, in remembering one's failures, that at one period of the disease the poison that is at the bottom of the condition is not sufficiently advanced, so that it is possible to overcome it by medicaments, but it is quite certain that at a later stage the disease overcomes you. So that as the disease advances there comes a time when everything which you do is equally unavailing. And it always does advance as far as I know, for I have never yet met with a case presenting the complete symptoms of the disease which was alive five years after the first attack.

I have brought down for your inspection two books which have in them very beautiful sets of plates showing the characteristic changes in regard to the red cells in this disease, Stengel's translation of Nothnagel's 'Encyclopædia' and the fourth volume of Osler and Macrae's 'System of Medicine.'

March 15th, 1909.

Atropin in Asthma.—Terray protests against the general neglect of atropin in treatment of bronchial asthma. As the affection is a neurosis, atropin is theoretically indicated, and he has witnessed excellent results from it in certain cases, although it is by no means a panacea in all cases. In one case he has succeeded not only in arresting the attacks with it, but the patient was improved so that there was no recurrence of the asthma for ten months.—*Journ. A.M.A.*, vol. lii, No. 9.

THE USE OF CONGESTION IN TREATMENT; ITS DISADVANTAGES IN VARICOSE VEINS, VARICOCELE, HERNIA, HÆMORRHOIDS, HYDROCELE, ETC.

A Lecture delivered at the Medical Graduates' College and Polyclinic.

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GENTLEMEN,—For many years now it has been possible to do a great deal to prevent suppuration, or the formation of pus as we had better call it. The prevention of pus has come to us most largely from the introduction of the antiseptics. But although much has been done to prevent the formation of pus, comparatively little has been done to treat the cases when pus has already formed. The time-honoured methods are by means of incisions and hot dressings. It has fallen to a German professor to teach us a clinical method of treating suppuration when already present by means which do not necessarily involve an incision or the use of hot dressings. This method of treatment has been termed "venous congestion," and I thought it might be useful as well as interesting to discuss with you the subject of the uses or advantages, and the dangers or disadvantages, of venous congestion.

The first point to discuss is the use or the uses of venous congestion. It is a method which is very easily applied. It tells quickly whether it is going to do good or whether it is useless. I particularly mention that this method may be useless because it has been taught, like all new things when they are introduced, that it is able to cure everything. You remember that when antitoxic serums were introduced it was stated in the lay press that there was to be no more disease, that there would be a serum for everything. Now we know that, with the exception of diphtheria, the various serums are of comparatively little therapeutic value. When Professor Bier introduced the use of venous congestion it was proclaimed that it was going to do everything. This method was applied to everything in Germany. They had special apparatus constructed to apply venous congestion to every region of the body, and even invented a kind of cradle in which they slung testicles for the treat-

ment of orchitis by venous congestion! You will see that in reality they were carrying too far what is a good method. Therefore I particularly point out to you that there are cases in which this method of treatment is useless.

I am only going to tell you how it may be produced in ordinary practice. It is not worth your while or my while to discuss with you the elaborate instruments constructed by means of which venous congestion can be produced on the back of the head or on the perinæum. In ordinary every-day practice one is able to produce venous congestion easily in two ways. First, in the limbs one can produce venous congestion by the simple means of taking a few turns of a common bandage or a rubber Martin's bandage round the upper arm or the thigh. Secondly, if the disease is situated too near the body for that, the venous congestion is produced by means such as our forefathers called dry-cupping. The glasses are like inverted egg-cups with a rubber squeeze-ball on the top, with which you pump air out. These are two methods you can employ to produce venous congestion in practice.

I was at St. Thomas's Hospital this afternoon and I mention two cases, one of which remains there under my care and one of which has just left the hospital, to show you that venous congestion produced by these two methods—by the bandage, and by the cup when the inflammation is on the trunk—is of value.

The first of those patients is a woman, æt. 35 years. When she came into the hospital she had her right elbow very much swollen: there was œdema and a superficial abscess. But the inflammation was more round the joint—peri-articular—than inside the elbow-joint. The left ankle was similarly affected; it was swollen, and the tissues about it were œdematous. When the ankle-joint is affected the sheaths of the tendons around soon become affected also. She had this tenosynovitis with painful swelling extending a little up the leg and down into the foot. Her temperature was 101° F. According to the old laws of surgical treatment the abscess in the arm should have been incised. The illness had been going on for a couple of months and obviously was not very acute, so that one utilised the presence of that disease to demonstrate to the students the value of the treatment by venous congestion. This woman had a bandage applied round the upper arm and thighs for two or three hours night and morning. It is now a fortnight since that was done, and her temperature has been normal for ten or twelve days. The abscess disappeared; it has never been opened. The swelling round the joints has disappeared also, and her elbow-joint can now be moved, revealing a lot of grating in it, and showing that there has been extensive disease. She is still in the hospital because the left ankle-joint is affected. Though this joint has the appearance of getting well the woman cannot stand on it. That is a simple case which anybody in practice could have treated in a similar way, and which required no particular intelligence or skill. The result is that the case has got well, whereas in former days she would have had the abscess opened, an incision on either side of the elbow-joint, and one on either side of the ankle-joint. Moreover, her convalescence would have been a matter of months. Instead of that the disease has been cured, a result requiring no special training to bring about.

The other case, who has left the hospital, was a man somewhere about 45 years of age admitted for a very severe condition called angina ludovici of the neck. In this disease you get a swelling in the submaxillary region which very rapidly travels down the neck and up to the jaw. The man when admitted was very ill. More than that, the cellulitis in his neck, besides extending to the clavicle, had gone inwards and he had marked stridor, due to œdema of the larynx. The man's pulse and general condition were such that it was decided to be unsafe to give him an anæsthetic. A comparatively small incision was made without an anæsthetic in order to relieve the tension in this cellulitic mass in his neck, and therefore the stridor caused by the œdema of his larynx, thereby enabling us subsequently, perhaps a day or two later, to give him an anæsthetic safely and incise the cellulitis freely and easily. We made this small incision in the middle of his neck without an anæsthetic, and as the neck is a place where you cannot apply a bandage very well to produce venous congestion we applied a cup such as you can buy from any instrument maker. By the aid of that cup we produced suction. The result was that his temperature came down, the stridor rapidly disappeared, the man improved in every way, and we never had any reason to make any

other incision. Furthermore, this man, who was admitted with angina ludovici, who was too ill to have a general anæsthetic, and who had stridor, was out of the hospital in fourteen days. That is another case whose treatment required no special training, and, as you see, the convalescence was quite a simple thing. These two cases show how by the use of venous congestion it is possible to produce in some instances results most beneficial to all concerned.

In connection with this case I may add that another man was admitted about the same time with a similar condition. He was not so ill as the other patient and we gave him an anæsthetic. The result of that anæsthetic was that the laryngeal obstruction prevented respiration. A patient with Ludwig's angina has a great bull neck, and it may be very difficult to do tracheotomy through three or four inches of tissue. When this man could not get breath, a rubber tube was passed through the larynx and he inspired freely. The cellulitis was incised. He is still in the hospital and not likely to be discharged for some time. On the other hand, as I told you, the man in whom we only made a small incision and applied the suction-glass left the hospital at the end of fourteen days. These cases are more or less parallel and show you that it is possible to reap very great advantages by the use of this simple method.

There are at least three points which you must remember in regard to the application of this method. Supposing you have a whitlow on the finger and use venous congestion for it, the *first* thing is that the pain must cease; it ought to cease within twenty minutes after the application of the bandage. That is an important point to remember because if the pain has not ceased the bandage is not applied rightly—perhaps it is too loose, perhaps too tight. In fact the bandage may need several re-applications to get the right tightness. But if you do get the right tightness and the case is going to be successful, the pain will cease within twenty minutes. That is an enormous score from both the doctor's and the patient's point of view, these cases having great pain and suffering from sleeplessness and loss of appetite. When the pain goes the patient sleeps probably for two hours; and when he wakes—the pain will still be away and the bandage will still be on—he will have a decent meal. So that the first point is that the pain must cease. With this cessation of pain the patient's temperature, which had been perhaps 102° F., or more, will fall. By the application of the bandage on the arm the toxins have been prevented from entering the patient's general system. Consequently, within an hour or two hours from the time of using the bandage his temperature may be normal.

The *second* point is that the arterial pulse on the distal side of your bandage for a whitlow just above the elbow must be unaffected, so that you only impede the return of the blood in the veins and of the lymph in the lymphatics. If you take care of that there need not be the least anxiety of anything like gangrene.

The *third* point in the application of this bandage is that the temperature of the limb, say of the forearm, must not fall. That you can ascertain easily. The patient by feeling it can say whether the arm is cold or not, and you yourself can easily compare the two forearms. The temperature must remain at the ordinary temperature.

If you pay attention to these three points—the cessation of pain, the arterial pulse and the temperature of the limb—you need not have any fear or worry about the application of this bandage.

The *rationale* of this treatment is that the bandage blocks the return of blood in the veins and of lymph in the lymphatics, producing some swelling of the arm. You will be disappointed if you expect to see much œdema as a result of the application of the bandage. You do not get much œdema. The swelling of the tissues below the bandage produces some infiltration of the connective tissue, with fluid, which has been compared to infiltration used to produce local anæsthesia, and is one of the reasons given why the pain goes. By preventing the return of the blood in the veins and the lymph in the lymphatics you get an aggregation of serum and leucocytes in the affected parts. The result is very valuable. We aggregate the patient's powers of defence in the diseased parts. We may illustrate it in this way. In 1900 the British Empire had a war in South Africa. That was the affected part, and we aggregated our soldiers there. In a similar manner this bandage aids materially in aggregating leucocytes, which are our main line of defence, in the affected part. But not only does the bandage cause the aggregation of leucocytes in the affected part, but it also causes the aggregation of serum

there. The use of passive congestion is that the defensive forces of the body are called to the part, like the response to the bugle. That is the *rationale* of this method of treatment, and that *rationale* I have gone into particularly because it will enable you to estimate the limitations of that treatment.

This treatment by venous congestion, like most other treatments, can *help* the body to recover, but it cannot *compel* the body to recover. One is apt to lose sight of that fact in the treatment of disease. We help to cure, we do not compel to cure. So that if you get a micro-organism introduced, we will say in a whitlow, which is so virulent that it is beyond the patient's leucocytes and serums to cope with it, no bandage is likely to be of any use at all. As I told you, it was thought at first that this treatment was going to cure everything, but the first thing found was that, if you have a cutaneous erysipelas the bandage is no good at all; the erysipelas will spread up the arm beyond the bandage. In some virulent infections, mostly with a streptococcus, we know that the bandage has failed.

More than that, it was supposed that this treatment was going to cure one of the most difficult things to treat—gonorrhœal rheumatism. This is an affection which takes months to cure. The patient may get some relief, but the disease goes on, the patient and the medical man becoming tired of each other. So far from curing gonorrhœal rheumatism, this method of venous congestion often makes the pain and the patient worse, so that one would not be keen on advising the use of congestion treatment for osteo-arthritis or gonorrhœal rheumatism.

There is another use for passive congestion. You know quite well those ulcers, more or less varicose, which women get on their legs and carry about for years. Some of those can be enormously benefited by means of these bandages. That was first thrust on me by a woman who was admitted under me at St. Thomas's Hospital to have her leg off. She wanted to have her leg off because she had had an ulcer so long and the heel had been drawn up so much that the toe was pointed in a position of marked talipes. At that time I was very keen on trying this method to see whether it would or would not do good. Before taking this woman's leg off, therefore, I tried the bandage. She had had this ulcer for many years, and we started congestion after having cleansed the ulcer first so that it was no longer septic. And I think I am correct in telling you that in nine days it was practically healed. Of course the woman was kept in bed during that time. I am talking of some four or five years ago. I have seen the woman two or three times since and it was all right. Whether it is all right at present I cannot say; you cannot follow hospital patients. But at any rate it was successful for a time, and so far as I know the success was permanent.

One would also mention that this bandage is very good for inflammation round a joint, rather than for inflammation in a joint. But it is no good for erysipelas, it is no good for deep cellulitis, it is no good for thrombosis or bone disease. That one knows definitely. It was cried by the German school who introduced this treatment that the knife was going to be banished from surgery. That is not true. One can make abscesses disappear as in the case I mentioned to you of the woman I have in St. Thomas's Hospital. But to do so is scarcely sound; it is somewhat of a gymnastic feat. It can be done, but it is very much better to make a small incision, quite a small one, or even to aspirate, and then apply venous congestion. So that if one is using venous congestion along with an abscess or cellulitis, instead of making a free incision which may take weeks to heal, make a small incision, even only a puncture, and then apply venous congestion. If you do that you will be more certain of getting a good result.

One may recount the clinical picture where this bandage is going to be successful. As I have said, the pain ceases, the patient feels relieved, the temperature falls, the pulse-rate declines, and the patient, who may have been absolutely sleepless, goes to sleep. The appetite, which had gone so that the patient was taking no nourishment, returns, so that when he wakes he will often take a good meal and feel a great deal better.

That is just a rough outline of the uses of venous congestion.

I want the remainder of the time to tell you of some of the disadvantages because of venous congestion. One can put the disadvantages or dangers, or whatever you like to call them, before you in a series of little short pictures. The first is in connection with *varicose veins*. The causation of varicose veins is going to be very greatly simplified. You will

remember perfectly learning how the valves of the veins were incompetent, how standing up caused the trouble, how varicose veins were attributed to back-pressure as it was called. That part of the causation is going largely to disappear. Varicose veins are due to a local disease of the vein-wall. As you can get a local disease in your big toe you can also get a local disease in the vein-wall. You get atheroma in your arteries: that is another local disease of the arteries. You can get also a local disease of the vein-wall. That is going to be the great fundamental groundwork of the causation of varicose veins. We know that quite well. For there is an operation, going by the name of Trendelenberg's operation, in which the internal saphenous vein is ligatured and a piece excised at the groin. That operation relieves the pressure on the diseased walls below where the vein has been tied. The result of the operation is very striking. In the great majority of cases the pain will cease, because by means of the operation you have stopped the back-pressure on the vein. But you have not stopped the veins from becoming varicose, new varicose veins often appearing. This shows that it is not the back-pressure that causes the varicose veins but local disease of the vein-walls. But the back-pressure is responsible for the pain. That back-pressure, venous congestion causing pain, is one of the disadvantages of chronic venous congestion.

Varicose veins have many complications, of which one may mention thrombosis, varicose ulcer, eczema, œdema. I do not want to waste time on any one of these except to dwell on eczema a little. We do not appreciate the fact very much that when people have varicose veins the return of the blood from the limb to the body is impeded and the limb is maintained in a condition of chronic venous congestion. We do not appreciate sufficiently that chronic venous congestion has a very important result on the skin. The skin may be thickened and more or less inflamed. On the other hand it may become thin, white and atrophied such as you see over some veins in the calf of the leg or in the scar of a gumma. I am leaving the subject of the affections of the skin as a result of chronic venous congestion.

As the second example of the disadvantages of venous congestion I will mention *varicocele*. You will remember the pathology. Your pathology taught you that the left testicle hung lower than the right, that the sigmoid might rest on the left spermatic veins, and Heaven knows what else. And so pathologists have tried to explain why varicocele is almost invariably on the left side. The pathology is going to be very much simplified. The fact that varicocele is on the left side is due to there being, congenitally, more veins in the pampiniform plexus on the left side than on the right. Varicocele is not only an enlargement of veins there, but a local disease of the vein-walls. And as in varicose veins, the pain in a varicocele is largely the result of back-pressure, venous congestion. Operation is going to relieve that pain, as by excising the varicocele the back-pressure is relieved, but it is not going to prevent the enlargement of other veins. The fact that the operation for varicocele—not, of course, in neurotic cases—relieves the pain is its justification, because it has no trace of justification as being beneficial to the testicle. A varicocele does patients harm, because the blood-flow returning from the testicle is slow. You do an operation and remove that varicocele, and the blood-stream is not slow; there is no blood-stream at all! You have removed the veins by which the blood should return, and by your operation you have produced a venous congestion of the testicle very many times worse than it was in the time of the varicocele.* It is from that point of view that the operation for varicocele is a foolish one; its use being that it relieves pain.

The next disadvantage of venous congestion is the wearing of *trusses for hernia*. It is obvious that a truss which presses on a hernia sac also presses on the spermatic veins and impedes the venous return within them. This does harm to the testicle, particularly in children, so much so that I never advise the use of any but the lightest trusses.

Another result of venous congestion is the *hydrocele of the elderly*. The male has a menopause similar to what the female has. That fact is very little appreciated. The date of the females' menopause we know quite well because it coincides with the cessation of menstruation. The time of the male "menopause" we do not know exactly, but there is an involution at that time; I would suggest that the period is somewhere about the age

* 'Diseases of the Male Generative Organs,' Oxford Medical Publications.

of fifty to sixty. That involution is accompanied by changes in the spermatic artery, which becomes thickened and its lumen almost blocked because the testicle which it is to supply no longer requires a larger amount of arterial blood for its nourishment. When the placenta disappears after birth you remember how the hypogastric arteries disappear altogether, becoming fibrous cords. It is somewhat similar with the spermatic artery after involution of the testicle. That is an important result, because the spermatic artery not only nourishes the testicle, but by it the blood is driven through the testicle along the spermatic veins to the heart. Block that artery and the blood in the veins is no longer urged onward as before, the tunica vaginalis and the testicle getting into a state of chronic venous congestion. This chronic venous congestion sooner or later means the chronic inflammation of the tunica vaginalis, and this again means a hydrocele. That condition of things is very likely the fundamental principle in the causation of hydrocele in the elderly.

There is one very interesting event which I must mention to you. You will remember, when I was speaking of varicose veins, I laid particular stress on the inflammatory and an atrophic action of chronic venous congestion on the skin. That knowledge is applicable to *hæmorrhoids*—varicose veins in the rectum—and has been thrust very much on me lately. A patient had been round to practically all the various spas and their medical authorities. He had a pain down the back of his legs and buttocks which was called fibrositis. He was sent to the spas, with the result that he did not get better. Finally they took to the plan of treating him with static electricity. At last somebody made a rectal examination, which caused great pain. When he came home I could not make a rectal or any examination on him because of the intense pain caused. I told him I would have to examine him under an anæsthetic, and did so. And now I will return to the action of chronic venous congestion on the skin. This man had had piles for upwards of twenty or thirty years. In other words the skin in the anal region had been in a state of chronic congestion, for that time, and the result was that it had become thin and more or less atrophic, so that as soon as the anus was dilated at all the skin cracked in one place or another. When dilated under an anæsthetic it split in several places. That used to happen no doubt when the man defæcated and there was the explanation of his acute pain. One removed as much of that atrophied skin and pile-bearing area as was possible and tried to fit him up again for a renewed lease of comfortable life. The result has been good. Although the case has only been done a little over three months it looks as though this pain was, in large part, referred pain from anal fissures, which were the result of chronic venous congestion of the anal region, from what we will call "neglected piles." I do not mean to assert that every person who has had piles gets this condition any more than all people who have had varicose veins get ulcers or atrophic shin. But it is quite certain that if people have varicose veins long enough a certain number of them do get varicose ulcers, and a certain number of them—not all—do get skin troubles. In a similar way some people with hæmorrhoids will get similar troubles. It has been very interesting to me that this trouble may appear, not under the surgeon but under the physician, as intractable pain down the back of the legs in connection with hæmorrhoids.

The points that I have brought before you are real points in the disadvantages of chronic venous congestion.

March 15th, 1909.

Pyocyanase in Diphtheria.—Grösz has used pyocyanase in the gravest forms of diphtheria and reports very favourable results. The best results were obtained by spraying the solution into the pharynx and even the larynx, for which purpose a special apparatus had to be constructed. In other cases inhalation of steam saturated with pyocyanase was tried. In all cases the pseudo-membrane disappeared very much more quickly than is usual in diphtheria, and the comfort of the patients was much increased. The temperature fell much more rapidly than with the use of antitoxin alone, and all general toxic symptoms were very favourably affected. In several severe cases of croup intubation was made unnecessary, the pyocyanase helping to separate the membrane very effectually; where intubation was done the tubes could be removed in much shorter time than has been usual.—*Med. Record*, vol. lxxv, No. 9.

A LECTURE
ON
TRIGEMINAL NEURALGIA AND ITS TREATMENT BY SCHLÖSSER'S METHOD.

Delivered at the Medical Graduates' College and Polyclinic.

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Of all the physical pains by which we may be attacked, that of *tic douloureux* is by far the most agonising. I do not propose to attempt to describe it at length; no words can give an adequate idea of its horrors. I would merely remind you that its outstanding features consist in paroxysms of exquisite pain in the area of one or more divisions of the trigeminal nerve, either the upper and middle divisions, or the middle and lower. The pain spreads in a definite order from one division to another; it rarely affects all three divisions. During the paroxysm of pain the eye on the affected side may brim over with tears, there may be excessive secretion of nasal mucus or of saliva, and in most cases the face on the affected side is contorted by tonic muscular spasm; hence the designation of "tic." These paroxysms of pain sometimes occur spontaneously. Sometimes they are excited by the slightest local stimulus, *e.g.* by the chewing of food, by pressure on the face, especially in the neighbourhood of the bony foramina of exit of the branches of the trigeminal nerve, sometimes even by a draught of cold air, by shaving, or by washing the face, so that it is not uncommon to find the affected side of the face begrimed with the dirt of weeks.

Tic douloureux is an inveterate malady which does not tend to spontaneous cure. On the contrary, its paroxysms tend to become more and more frequent; all drugs, short of morphia, fail to give relief, and the patient either becomes a morphia *habitué*, or at last he may be driven to suicide. Its treatment, therefore, may become a matter not merely of the patient's comfort, but actually of life and death.

Before regarding any individual case as one of true *tic douloureux*, we must exclude the minor degrees of neuralgia in the territory of the trigeminal nerve. We must search for and remove all possible peripheral causes of reflex pain in the mouth, the nose and its accessory sinuses, the ear, and the eye. We must also exclude the possibility of gross organic disease at the base of the skull, whether tumour or gummatous infiltration, compressing or invading the trigeminal nerve or its branches at their foramina of exit. Such cases of gross organic disease usually produce other signs and symptoms in addition to pain, *e.g.* signs of affection of the motor root of the trigeminal, or a degree of anæsthesia in the painful area, or affection of other cranial nerves. In pure *tic douloureux* there is neither motor weakness of the muscles of mastication nor cutaneous anæsthesia; on the contrary, there is often exquisite hyperæsthesia of the skin and mucous membranes in the affected area.

Microscopic examination of the Gasserian ganglion and of the branches of the fifth nerve in these cases reveals no recognisable pathological changes, so that we are completely in the dark as to its true causation. All that we can say is that the definite localisation of the pain, in the first instance, to one and afterwards to other divisions of the nerve, points to the disease as arising from a cause distal to the Gasserian ganglion.

Attempts at relief of the disease have taxed the ingenuity of countless physicians and surgeons, First, as to drugs, their name is legion. But in early cases it is our duty to give medicinal treatment a fair trial. Innumerable analgesic remedies have been employed. Some of these are applied locally, for example, aconitine ointment, liniment of mesotan or menthol rubbed into the face; or again, we may try ionic medication with salicylates or iodides by means of a galvanic current. Leeches and local counter-irritants such as mustard or cantharides have been used. Internally analgesic drugs of all sorts have been tried, of which, perhaps, the most familiar are butyl chloral hydrate, phenazonum, and acetanilide. Sometimes a prolonged course of gelsemium in full doses, or of arsenic, seems to diminish the hyperexcitability of the nerve. One or other of these remedies may for a time alleviate the patient's suffering, but, sooner or later, in a severe case, drug treatment fails and we are left with the two alternatives of chronic morphine administration or of some operative measure. All of

us will agree as to the grave dangers of allowing a patient to establish the morphia habit. We are, therefore, finally driven to attack the nerve itself. The object of any local attack on the nerve is to interrupt the conduction of sensory impressions from the painful area. Minor operations have been tried, such as division, or avulsion, or injection of osmic acid into the peripheral branches of the nerve, and with varying success in affording temporary relief. Thus the infra-orbital nerve has been attacked in its bony canal, the supra-orbital nerve reached through its notch or foramen, and the inferior maxillary division approached either at the mental foramen in the chin or further back through a trephine hole in the ramus of the lower jaw. These peripheral operations sometimes give relief for a few months, but ultimately the pain returns and we are forced to attack the nerve closer to the brain, in the region of the Gasserian ganglion itself. At one time the ganglion was removed extracranially by means of a difficult operation (Rose's) in the pterygo-maxillary region. Nowadays, however, surgeons prefer the operation of Krause or of Abbe, in which the ganglion is removed by the intra-cranial route, being lifted from its bed at the front part of the petrous bone. The motor root of the trigeminal is usually sacrificed along with the sensory ganglion. This intra-cranial excision of the Gasserian ganglion has been performed in a very large number of cases in this country by such surgeons as Horsley, Ballance, Hutchinson, and others, and with results infinitely superior to any previous method of treatment, so far as relief from pain is concerned. But removal of the Gasserian ganglion is a formidable operation, associated with a considerable mortality, and even when successful the paralysis of the trigeminal nerve which necessarily follows has a number of inconvenient results, including anæthesia of the corresponding side of the face, conjunctiva, nose, mouth, tongue and teeth; also paralysis and atrophy of the masseter, pterygoid and temporal muscles, producing clumsiness in mastication. Sometimes also there occurs a so-called "neuro-paralytic" keratitis, which Davies and Hall have shown to be due to infection of the conjunctiva with a special bacillus. All these are grave inconveniencies; yet the patient is willing to risk them all, rather than suffer a continuance of his agonising pain. It must also be mentioned that occasionally the pain recurs, even after excision of the Gasserian ganglion. I have myself seen instances of this. The explanation of such cases is obscure. Some have attributed them to regeneration of the divided nerve-fibres, and with the object of preventing such regeneration they plug the foramina of exit of the nerves with gutta-percha or with gold-leaf. Excision of the superior cervical ganglion of the sympathetic chain has also been tried by some surgeons with the object of producing a vaso-motor paralysis on the affected side, but I cannot speak from personal knowledge of the results of this operation.

The latest method of treatment, to which I propose to direct your attention this afternoon, is that devised a couple of years ago by Schlösser, of Munich.* This consists in the injection of alcohol into the affected nerve-trunks at their foramina of exit from the base of the skull, close to their emergence from the Gasserian ganglion. Injections such as these produce temporary pain for a few minutes, which may be largely mitigated by the addition of a small amount of cocaine to the alcohol. This pain is followed by anæsthesia for several days of the affected nerve-area, together with freedom from the old pain. Though the anæsthesia passes off, the pain does not return, and the period of relief after such injections, according to the statistics of 123 cases treated by Schlösser, averages between ten and eleven months, and is sometimes much longer. The injections can then be repeated. Each relapse is milder than the previous attacks and the intervals become longer. Kiliani,† of New York, has published a large number of cases treated by the injection of 80 per cent. alcohol, whilst Patrick and Hecht,‡ of Chicago, prefer a solution of 75 per cent. alcohol containing cocaine. Their formula, which is the one I have myself employed, is as follows: Cocainæ hydrochlor., 1 gr.; chloroformi, 10 ♏; alcohol absolut., 3 drachms; aq destill to ½ oz. Of this solution 2 cubic centimetres are injected at a time.

The technique of the injection is as follows: A stout needle, 10 cm. long and 1·5 mm. in diameter, is used. Within this is fitted a blunt stilette whose point projects slightly beyond the sharp end of the needle. The needle is marked off in centimetres up to 5 cm., so that we

* 'Berlin. klin. Wochenschrift.,' 1906, No. 3.

† 'Medical Record,' January 18th, 1908.

‡ 'Journ. Amer. Med. Assoc.,' November 9th, 1907.

may know at what depth the point is. In making the injection through the skin (previously disinfected, and frozen by chloride of ethyl) the stilette is slightly withdrawn, so that the sharp point of the needle pierces the skin easily. When the point is through the skin, the blunt stilette is pushed home; thus the rest of the penetration is done with a blunt-ended instrument and no injury is done to the deep vessels. When the end of the stilette has reached the foramen in the skull, at the proper depth, the stilette is completely withdrawn; a glass syringe, ready filled with the solution, is fitted to the needle, and the injection is slowly made. We leave the needle *in situ* for a couple of minutes and then withdraw it. There is usually no bleeding, or, if any, it is so trifling that it is quickly stopped by pressure. The needle puncture is covered with collodion and the whole affair is completed within a few minutes. A general anæsthetic is not required; in fact, we prefer not to use one, for when the injection is successfully made into the nerve-trunk the patient feels a burning pain along the distribution of the nerve which shows we have reached the right spot. If cocaine be added to the alcohol, this pain passes off very soon, and is followed by anæsthesia as already described.

Certain minor complications may be associated with injections of this sort. For example, we may have slight œdema of the eyelids on the affected side after injections into the upper or middle divisions of the nerve, whilst after an injection into the third division the patient may complain of some stiffness in the masseter and pterygoid muscles. In a few cases temporary paresis of the facial muscles, lasting a few days, may occur after injection into the second division of the trigeminal. Kiliani has suggested that this may be due to the connection between the facial nerve and the spheno-palatine ganglion *viâ* the Vidian nerve.

To reach the foramen ovale, through which the third division of the trigeminal emerges from the skull, the needle is inserted through the cheek behind the last upper molar tooth at the lower border of the zygoma,* at a point 2½ cm. in front of the descending root of the zygoma, which can always be felt as a ridge close to the anterior border of the external auditory meatus. After piercing the skin, we push home the blunt stilette and proceed to penetrate the deep tissues, comprising the masseter and the posterior part of the temporal muscle. The needle is directed backwards and slightly upwards till it impinges on the skull at the external pterygoid plate. It is then pushed on, upwards and backwards along the bone, until it comes to the base of the pterygoid process. It is then pushed backwards and slightly more upwards until it enters the foramen ovale, at a depth usually of about 4 cm. from the zygoma. The stilette is now withdrawn, and the injection is slowly made. An occasional difficulty to which other observers have not referred, but which I have occasionally met with, may occur in edentulous subjects, in whom the whole mandible is displaced upwards, so that the upper part of the ramus and coronoid process are overlapped by the zygoma, thereby preventing the needle from reaching the deeper structures. This difficulty is easily overcome by making the patient open the mouth widely.

To reach the foramen rotundum, through which the second division emerges, is less difficult. Here our guide is the posterior border of the orbital process of the malar bone. We prolong this line downwards to the lower border of the zygoma and insert our needle ½ cm. posterior to this point. Pushing horizontally inwards, and directing the point slightly upwards, the foramen rotundum is reached in the pterygo-maxillary fossa at a depth of about 5 cm. from the zygoma, at a level with the inferior extremity of the nasal bones. The structures pierced by the needle are the anterior fibres of the masseter and the anterior edge of the temporal tendon, though this latter may be missed if the patient opens the mouth widely.

The supra-orbital division, emerging through the sphenoidal fissure at the back of the orbit, is rather more difficult to reach with certainty, owing to the proximity of the optic nerve and of the third, fourth and sixth nerves. Patrick and Hecht admit that the nerve is more often missed than reached by the needle, but it is not essential that the nerve-trunk should actually be touched (though, of course, we should try and do so), because if the alcohol be injected in the immediate neighbourhood of the nerve it diffuses into it. The needle is inserted just at the outer margin of the orbit, close within the fronto-malar articulation, and is

* Levy and Baudouin, 'La Presse Médicale,' February 17th, 1906.

passed along the floor of the orbit to a depth of $3\frac{1}{2}$ or 4 cm.

Although a single injection may suffice to relieve the pain, it is advisable to repeat the injection after a couple of days, and until the last trace of pain disappears we repeat the injections at short intervals. In one of my cases no fewer than seven injections were required before the patient was finally relieved.

Let me mention a few illustrative cases:

Case 1.—A male patient, æt. 60 years, had suffered for ten years from paroxysmal pain in the second and third divisions of the right trigeminal nerve. The pain was accompanied by the usual spasm of the face and by lacrimation of the right eye. The attacks were precipitated by attempts at mastication, also by emotional excitement. This patient had seven injections in all, introduced into the foramen rotundum and foramen ovale, at first at intervals of a day or two, later at intervals of a week. After the second injection the painful paroxyms were reduced to only three or four momentary "flashes" *per diem*. Later these ceased also, and he is now, after four months, free from all pain.

Case 2.—A male patient, æt. 69 years, had suffered from *tic douloureux*, chiefly in the third division of the trigeminal, for seven years. The pain had progressively increased in severity in spite of various drugs by the mouth and hypodermic injections of cocaine. All the teeth, upper and lower, had been extracted on the affected side without avail. The patient was in constant agony, night and day, walking about striking his head against the wall. He was unable to masticate and had to be fed with fluids. He came up to London with the object of having the Gasserian ganglion removed. But being a stout man with a feeble heart, albuminuria and œdema of the legs, Mr. Ballance, to whom he had come for operation, wisely deemed the risk too great and accordingly he was seen by me. Injections of alcohol and cocaine were performed into the foramen ovale and foramen rotundum. These were repeated four times in all within ten days. After the first injection the patient called for his teeth and ate a mutton chop, the first for years. The pain recurred a week after the first injection, but by the end of twelve days (the fourth injection having been done on the tenth day), the pain subsided. Since then, nearly five months ago, the patient has remained free from pain.

Case 3.—A male patient, æt. 46 years, had suffered for three years from severe pain in the right side of the face, particularly around the eye, on the scalp and down the side of the nose, in the distribution of the first and second divisions of the nerve. The paroxysms steadily increased in severity and for six months he had been unable to chew solid food. He kept his face very rigid when speaking, in order to avoid all facial movement, which excited the pain at once. He had been treated by various analgesic drugs without result.

Three injections in all were given into the foramen rotundum, the second two days after the first. Immediately after the first injection the spontaneous paroxysms of pain ceased, but he could still induce an attack if he wiped his upper lip. A third injection was given on the seventh day. Since then, four months ago, he has been entirely free from pain.

Case 4.—A woman, æt. 56 years, had suffered for three years from paroxysms of pain in the right temporal region, extending into the right upper gum. These paroxysms were excited chiefly by washing the face, and she was unable to wear her artificial teeth owing to the excitation of pain by the attempt at chewing. She was assiduously treated by arsenic and gelsemium, also by various analgesic drugs by myself for over two years. Osmic acid was then injected into the infra-orbital foramen and this relieved her for two months, when the pain recurred as badly as ever. An injection of alcohol and cocaine was then made into the foramen rotundum. Since then, three months ago, she has been entirely free from pain.

I have only been employing this treatment myself for some five months, so that I merely quote a few of my early cases, and am not yet in a position to state the duration of the relief from pain. The cases I have mentioned, however, are fairly typical.

All of them were of such severity as to justify excision of the Gasserian ganglion, but in one at least (Case 2) the patient's general condition was such that the risk to life of such an operation was too great. Yet in this case Schlösser's method produced relief. With a knowledge of anatomy, and after some practice, the injection can be done with comparative ease. I myself perform it in the out-patient department, no general anæsthetic being required. The only part of the puncture which is painful is the penetration of the skin. This pain, as we have seen, is obviated by freezing with ethyl chloride. When we reach the nerve-trunk a second temporary pain is produced, which is transient. Moreover, no deformity or paralysis result such as of necessity follow excision of the Gasserian ganglion. We have therefore a valuable method of treating a class of case, which is often the despair of the most accomplished therapeutist.

March 15th, 1909.

ALCOHOL IN MEDICINE.

We have received from Messrs. Cassell & Co. a copy of the new edition of "A Manual of Medical Treatment," by I. Burney Yeo, M.D., F.R.C.P., Raymond Crawfurd, M.A., M.D., F.R.C.P., and F. Farquhar Buzzard, M.A., M.D., F.R.C.P. This work has gained such an established prestige that the appearance of a new edition strikes one as a matter of course, and, as might be expected, the process of thorough revision which the work has undergone has kept it well abreast of modern medical progress, especially in regard to actual clinical work. The practical application of recent valuable laboratory researches on the preparation and investigation of anti-bacterial and antitoxic sera and of protective vaccines is discussed fairly and frankly. At the present time, when the question of alcohol in medicine is exciting so much attention, it is interesting to note the views of the authors of this work as shown in the following passage:

A most important question in connection with the management of the gouty is the use of alcoholic drinks. In most persons, and especially in women, it is best to avoid them altogether; in others their moderate and discriminating use is not injurious. Of all alcoholic beverages malt liquors are most prejudicial in this diathesis, and heavy, bad-quality wines. With regard to wines, it is not the name but the quality of the wine which is important; and a matured wine of fine quality will often be found to agree with a patient who would be made ill by the same wine when of inferior quality. The wines which act freely as diuretics are the wines which agree best with all persons. In some this will be champagne, in others claret, in others hock, and so on. A very eminent physician of New York told us that he found champagne and port—carefully selected—the best remedies for *his* gout. We have certainly known many gouty persons who found champagne the wine that suited them best. But it will also happen that gouty persons who have drunk champagne with impunity for many years will suddenly find that it provokes some form of goutiness which disappears on relinquishing the use of this wine. For those who find a certain amount of wine a necessity, it is advisable that they should add to the wine they drink a small quantity of an alkaline water. In the case of some feeble gouty patients we have found a glass of sound port, matured in *wood* and diluted with twice as much *hot* water, a harmless and comforting drink, especially in winter. *Still* Moselle mixed with Apollinaris water is a good summer drink for thirsty persons who require a "big drink." For others two or three table-spoonfuls of old whisky or good cognac daily freely diluted with water may be permitted. But it is undoubtedly better for the gouty person to abstain entirely from alcohol if he can do so with comfort.

REMARKS UPON POPLITEAL ANEURYSM.

By EDMUND OWEN, D.Sc., F.R.C.S.,
Consulting Surgeon to St. Mary's Hospital, London.

The moral of Mr. Bowlby's interesting lecture in the Clinical Journal of March 10th, 1909, is that because a man has a popliteal aneurysm it by no means follows that he is to be operated on. I would like to support this contention by giving a brief report of a case in point.

About twenty years ago, more or less, a Fellow of the College of Surgeons in general practice came to consult me about a varicose condition of the external saphenous vein which had recently made its appearance. The dilatation was due to the pressure of a large popliteal aneurysm, of the existence of which he had no knowledge. His radial and femoral pulses showed considerable arterial tension, and his heart's action was excited and tumultuous. I sent him home to bed and told him that I would come and see him early in the afternoon. He was a strong man of athletic build, and of about 65 years of age. I felt sure that if an operation was done for his aneurysm he would have died. Mr. Savory, being a friend of his, kindly came and saw him with me and confirmed my opinion that no operation should be advised, as he seemed to be the subject of general arterial disease. We therefore contented ourselves with raising the foot of the bed, so as to help the venous return, and with laying the limb upon a pillow. So far as I remember the only medicine prescribed was calomel. Nevertheless the aneurysm increased in size, and we arranged that in case of its bursting or of mortification setting in we would amputate in the lower third of the thigh. But neither contingency arose, though the foot and leg became cold and pulseless. The aneurysm eventually grew still and solid. After he had been lying in bed about ten days the other popliteal artery became aneurysmal. This we treated by placing a firm pad at the back of the knee and keeping the leg firmly flexed. Professor Humphry, who also was a great personal friend of the patient, used to see him with Mr. Savory and me from time to time. He was fully in accord with the line of treatment by "masterly inactivity." In the course of a few days the second popliteal artery became solid like the first, and pulsation disappeared in the arteries at the ankle. He made a complete recovery, and a few years afterwards I saw him running after an omnibus.

March 15th, 1909.

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DYSPEPSIA DUE TO ALCOHOLIC ABUSE.

A Lecture delivered at the London School of Clinical Medicine.

By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Consulting Physician to St. Bartholomew's Hospital; Senior Physician to the Seamen's Hospital, Greenwich.

GENTLEMEN,—I ask you to consider with me to-day the nature and treatment of cases of dyspepsia which are dependent on the misuse of alcoholic drinks.

We may note first that all the symptoms of this condition are probably not due to the harmful effects of alcohol alone, for we must bear in mind that in many of these patients there has been more or less inanition due to loss of appetite, and that aggravation of the dyspepsia is not seldom the result of drinking bad liquor.

The condition is simply dependent on acute gastritis or gastro-enteritis, which ensues most commonly on a debauch or bout of drinking various alcoholic liquors lasting for a few days. These sad outbreaks, as we know too well, are somewhat apt to occur in sailors or soldiers, and in other classes, too, on their liberation from ordinary discipline and duties, and at some particular seasons such as Christmas time and the beginning of a new year. It is right to note that such lapses of morality are, happily, becoming less common than they were. In other instances we find that men and women break out at intervals, sometimes long and sometimes short, with attacks of dipsomania or active drinking, and become the subjects of the particular dyspepsia we are considering to-day. These persons, however, are the subjects of a form of neurotic instability or insanity, pitiable enough, because they are irresponsible for their conduct,

and are otherwise, not seldom, estimable and quite exemplary people.

The results of a drinking bout are manifested after a varying period, commonly of a few days, by a state of stupor and exhaustion, with a tendency to sleep. The excitement stages of alcoholism may have passed off, and we are not concerned with delirium, tremors, or delusions, which represent another phase of debauchery. I will only remind you in passing that the latter symptoms are more apt to occur in persons of superior mental power, in the clever and accomplished rather than in dull, stupid, and bucolic individuals.

We may note pallor of the face, cold perspiring skin and a feeble soft pulse, and a normal or slightly raised temperature. There may be pain in the epigastrium, hiccough, nausea, retching and vomiting of mucus or blood and mucus. The appetite for ordinary food is lost. The tongue is thickly furred and the breath foul, with a mawkish and possibly alcoholic odour. A severe headache is common, with a sense of fulness and oppression. There may be a craving for spiced and strongly flavoured articles of food, and a desire for further potations of some alcoholic liquid.

The bowels are confined, and the urine is scanty and loaded with lithates. In some cases the pharynx and œsophagus partake in the irritated condition of the stomach and intestines, and lead to absolute inability to swallow either solids or liquids, a mouthful of liquid being at once rejected. I remember one of the old sisters at St. Bartholomew's Hospital telling me that such cases used to occur about Christmas time in the meat-carriers of Smithfield Market. They are not met with now. A minor degree of this pharyngeal irritability is, however, very often present in drunkards, giving rise to the morning retchings.

There are not often opportunities of ascertaining the precise state of the gastric and enteric mucosa in these cases, for they commonly recover under suitable treatment. An attack of pneumonia is the most frequent cause of death unless the dosage of alcohol has proved *absolutely* toxic to a system already impaired in vigour, and depressed by deprivation of proper food and exposure to inclement weather. We may feel sure that we have to deal with acute gastritis or intense catarrh of the mucosa, and the appearances we know are then distinctly of an inflammatory state, indicated by engorgement of vessels, aphthous patches, exudation of grumous blood, and copious outpouring of mucus. The tubal glands display degeneration of their epithelial cells, with free desquamation of them. The submucous tissue is œdematous and choked with leucocytes. That is the morbid anatomy. The liver may be engorged and tender on pressure. If the process extends beyond the pylorus we may expect the occurrence of diarrhœa. This is not infrequent in topers, and is known as *diarrhœa crapulosa.* Sometimes the condition of phlegmonous gastritis has been met with.

These, then, are the symptoms and disordered elements we have to deal with in the graver cases of abuse of alcohol which constitute a *debauch.* In more chronic forms we meet with an abiding dyspepsia which is more frequent, though void of the more urgent symptoms just described. Gastric ulceration is apt to occur in some of these cases, especially in elderly men, and to give rise to severe pain and sometimes to copious and even fatal hæmorrhage, particularly when the splenic artery is eroded by the ulcerative process. There are also present obvious degenerative conditions in the arteries, liver, and often in the kidneys.

It is well for such patients if they secure the benefit of hospital treatment, and so avoid a continuance of alcoholic dosage which they and their friends are too apt to recommend by way of cure.

The first point in treatment is to cut off all alcohol. If the œsophagismus or spasm of the irritable pharynx and gullet is so great that no form of nourishment can be taken, we may reduce this by the use of a spray of solution of cocaine, 2 per cent. strength, and if some of this is swallowed the action may be still more beneficial. Vomiting may be usefully encouraged when swallowing is possible by draughts of warm (not hot) water, and these help to cleanse the mucous surfaces of foul exudation. Whey is the next article of nourishment to employ, and may be given in any quantity, followed up by milk containing a drachm of sodium bicarbonate to the pint, or combined with equal amounts of well aërated soda-water. A large linseed and mustard poultice is to be applied to the epigastrium, and repeated, or not, according to the tolerance of the skin for it. If diarrhœa is persistent lime-water should be added to the milk, and castor oil with tincture of opium given. Half-grain doses of calomel repeated

every half hour may be of service in this condition, or a full dose of gr. v or gr. x given overnight followed by a draught of mistura alba the next morning. Continued vomiting may require doses of ℥iv of dilute hydrocyanic acid in effervescing soda-water, and a mixture of subnitrate of bismuth with sodium bicarbonate may next be given. The dietary is next to be mended by the use of jelly of mutton or veal essence, and, later, pounded fish may be given. As the digestion is restored, it is found useful to give a bitter tonic containing several vegetable bitters, such as calumba, gentian, with five or ten minims of tincture of capsicum, and if there is pain a few minims of tincture of opium may be given with each dose. It is important to mention here that some experienced topers have long found the value of such a mixture as this, and recognise the special benefits of the opium in it, and this to such an extent as to cease their alcoholic potations and drift into opium takers. This has been observed especially in the case of women. This resort to opium and other stupeficants has been largely observed in the United States, in such parts as have come under strict legislation with regard to alcoholic habits, illustrating the folly of such repressive measures, and the impossibility of mending bad habits by improper means.

It is remarkable how fully such sufferers as I have described recover entirely from the serious effects of their foolish indulgence. Especially is this the case in young men. We witness this in sailors, who return to their duties and maintain robust health when leading strenuous lives under discipline, and away from the temptations and curses of their environments ashore. As I have said, we now see less of this gross debauchery in this worthy class of men, thanks to the better efforts which are made to help them to better ways, to more rapid voyages, to better dietary at sea, and more care for their highest interests. We recognise all this, but still feel assured that there is yet much more to be done to help these men and many others to learn and to practise self-control. All evils mend in this world, but some mend slowly. Violent legislation will not mend them, but a persistent and brotherly interest in our fellow-men will assuredly not fail to help the tempted and erring ones amongst us.

March 22nd, 1909.

ATHLETICS IN RELATION TO DISEASE.*

By R. H. A. WHITELOCKE, F.R.C.S.Eng.,

AND DISCUSSION THEREON BY

DR. COLLIER. DR. DE HAVILLAND HALL.
SIR LAUDER BRUNTON. DR. VOELCKER.
DR. SAMUEL WEST. MR. LOCKWOOD.

Mr. R. H. A. WHITELOCKE (Oxford), in opening the discussion, said: I wish in the first place to express my sincere thanks to the Fellows of this Society for having invited me to open the discussion this evening. More especially do I feel it a high honour inasmuch as I am a total stranger and have not even the privilege of Fellowship. The subject is one which has always been of special interest to me, for during the last twenty years I have lived and practised within the precincts of one of our oldest Universities and have acted as Surgeon to the football and athletic clubs at Oxford. Most of you must be fully aware of the important part that athletics play in the daily life of the junior members of our Universities. Many opportunities have been afforded me for noting the close relationship between athletic exercises and injury and disease. It would be quite out of the question for me to attempt to give you anything like a full account of the many and varied disabilities which from time to time are met with. The time at our disposal is too short; I will therefore confine myself to bringing to your notice some of those which appear to me as most profitable for discussion. Violent athletic exercises produce an effect upon many of the important internal organs of the body, such as the lungs, heart, kidneys, brain, and digestive organs, and give rise to symptoms which may be even alarming. One of the first effects of violent exercise is an increase in the carbonic acid in the blood. To eliminate this the action of the heart is increased so as to drive more blood into the lungs for it to be there oxygenated. Breathlessness soon follows, for the increased amount of blood taken to the air-vesicles of the lungs actually diminishes the amount of air contained in these organs at any one time. The effect of this change is that increased inspiratory efforts are made to

* Delivered before the Medical Society of London.

compensate for the loss of air space. This can only be brought about by a fuller distension of some of the vesicles, which up to this time had been only partially distended or inflated. The parts of the lungs in which this change takes place most are the anterior and posterior borders and the apices. Thus a kind of physiological emphysema is produced, and it is possible that when the proper balance between the blood supply and the lung distension is effected that the phenomenon known as "second wind" is accounted for. It becomes a question whether this physiological emphysema in the course of a strenuous athletic life may not over-reach its proper bounds and become pathological. If an examination is made of the chests of well-known athletes it is not unusual to find a considerable amount of this emphysema. It is so frequently found in persons who appear to be in the very best possible health, and whose athletic prowess is very great, that one is constrained to regard the change as physiological and as beneficial rather than harmful to the individual.

Heart.—The heart, like every other muscle in the body, may be over-strained as the result of violent athletic efforts. Such a state of affairs is much more likely to occur to ill-developed, under-fed and delicate youths of the lower class in our large cities than to University undergraduates, whose conditions of life are more favourable. Nevertheless, heart-strain is not unknown amongst the members of our Universities. The first effect upon a weakened heart of violent and sustained efforts is dilatation. The right ventricle is the chamber most usually affected in the first instance, though the auricle also suffers, and gives rise to a train of symptoms which are more or less characteristic. The patient comes complaining of palpitation, breathlessness, giddiness, and inability to endure any sustained effort, and often troublesome cough. On making an examination of such a case the epigastric pulsation at once attracts notice. Percussion reveals an enlargement, chiefly of the right heart, and auscultation a systolic bruit which is heard for the most part along the left border of the sternum. The prognosis of these cases is almost invariably good, for if the individual can be persuaded to give up all strenuous games, and to go in for gentle out-door exercise in the fresh air and to carefully observe moderation in all things, improvement may be expected in a short time. Treatment consists in prohibiting all violent and sustained efforts, and recommending a carefully regulated life. Arsenic combined with strychnia makes a good tonic: the arsenic seems to give tone to the muscle-fibres and the strychnia braces the nervous system. Athletic exercises, if strenuous and long continued, give rise to *hypertrophy* of the heart, as evinced by the displacement of the apex beat downwards and to the left and by the slow vigorous pulse. This hypertrophy must, in a majority of cases, be looked upon as physiological. It is only when athletics are pursued continuously for some considerable time—it may be even for years—that this form of enlargement is to be looked upon with apprehension. Men under forty rarely suffer from any inconvenient symptoms as a result of it, and as athleticism is seldom continued up to this age real harm is not often done. The one symptom which, as far as my experience goes, is most complained of by old athletes, is insomnia, and this seems to be caused by cardiac hypertrophy. Those who have suffered early in life from heart-strain seem to show a distinct tendency to recurrences in older life when mountaineering and other violent exercises are attempted without due and gradual preparation.

Effect on the kidneys.—That the kidneys are also affected is certain, for a definite though transitory and functional albuminuria is very frequently occasioned by violent exercise. This subject of intermittent albuminuria has been so fully and so well introduced to this Society in recent times by my friend and colleague Dr. William Collier that there remains but little for me to add. Those of you who may not have seen his paper I think will do well to read it now. It was published in the 'Transactions' of this Society as well as in the 'British Medical Journal.' That writer demonstrated that as a result of his own personal investigation the greater number of athletes who went in for severe competitive athletic contests were the subjects of albuminuria. The effect was the same whether the individuals ran, rowed, marched, or played at football. I may say that the same has been my experience whenever I have had occasion to test the urine of my young athletic friends after they have been competing, and I am perfectly in agreement with most of what Dr. Collier has written on this subject. In a few cases I made a microscopical examination of the urinal deposit after the

secretion had been allowed to stand for some hours. Amongst other things I found spermatozoa and blood-cells, both red and white. This has led me to believe that a certain amount of the albumen found after violent exercise may be due to the presence of seminal secretions. Nor is it difficult to understand why this should be present if we for a moment consider that we are dealing with healthy individuals in the full vigour of early manhood, who are not only secreting freely on the one hand but are also on the other liable to expel these secretions mechanically during their muscular exercises. The rowing man probably exerts pressure on his vesiculæ seminales in his efforts, as does the long distance runner in his steady jogging stride. During the act of defæcation what is regarded as a physiological emptying of these receptacles often occurs, and the more continent the man the more frequently does it seem to appear. The presence of blood may be quite accidental, but it should be noted, as it may become a matter of much importance. I had a patient who, after every game of Rugby football, showed traces of hæmaturia. At my advice he gave up football and took to riding; the hæmaturia became more and more intermittent and in about six weeks ceased altogether. One day he was asked to make up the number of a team to go down to his old school to play football. He played much against his will, and unfortunately suffered from a severe attack of hæmaturia a few hours after the game. The question of prognosis is a very important one and was dealt with by Dr. Collier and discussed by the Fellows of this Society. My experience, as far as it goes, coincides with the belief then expressed that these albuminuric persons soon lose all symptoms shortly after they give up violent exercises. The prognosis may therefore be said to be good. Medical treatment seems to have little or no direct effect on the quantity of albumen excreted. It may be that these symptoms of albuminuria and hæmaturia are the result of some increase of blood-pressure in the kidneys, or that the organs acquire some ill habit, and I should much like to hear in the discussion this evening the opinions of those present as to a definite theory of the exact ætiology as well as the views generally held as to the acceptance or rejection of candidates for the public services who exhibit these transient and intermittent symptoms.

Various symptoms of digestive disorders occur; acute attacks of indigestion, loss of appetite, and even catarrhal jaundice are not unknown in persons who over-train. Nervous symptoms, such as irritability, depression, insomnia, and inability to do strenuous mental work are sometimes present. "Getting stale" is due to a form of mental monotony. All these disabilities really represent symptoms rather than diseases, and they are fortunately functional and transient. They all seem to disappear more readily by the discontinuance of violent exercise than by any system of medicinal treatment.

Injuries to the internal organs. — Injuries to internal organs occur, and may be very serious. The viscera most frequently injured are the brain, spinal cord, kidneys, and eyes.

Concussion of the brain is by no means rare, and varies considerably in degree; sometimes there is a very short period of unconsciousness followed by some slight mental confusion, with perhaps sickness and headache the next day. In more serious cases the unconsciousness lasts for hours. I have known temporary mania follow upon a head injury that was at first quite slight. Such cases are very rare. It is a wise precaution to take, nevertheless, that a concussed patient should be kept quiet for at least eight days, all exertions, mental and physical, being prevented.

Spinal symptoms sometimes occur. Tingling and numbness in the extremities are often experienced, and even temporary paraplegia has followed upon acute and violent flexions of the spine in the cervical and upper dorsal regions.

The kidneys are often bruised, as evinced by the transient hæmaturia which follows upon a blow in the loin or a severe squeeze while playing at Rugby Football. It usually lasts for about twenty-four hours, and the blood varies much in quantity. Renal colic may follow from the passage of clots down the ureter. The treatment is rest and attention to general principles. On three occasions I have met with laceration of the right kidney, in each case the result of a kick while playing Association Football.

One case came to my house to report himself, fainted, and nearly died on the floor of my room from internal hæmorrhage. For many hours he hung between life and death, and the symptoms were relieved by the use of ergotin and extract of suprarenal capsule. After a long convalescence he recovered.

In another case, although the shock was not so great, an extravasation of urine followed, which was treated by operation and drainage. He ultimately recovered, and is now an officer in the Army.

The testis is often injured. On one occasion I saw a partial detachment of the retina from the blow of a football on the right orbit, and on another a dislocation of the crystalline lens from the same cause. These are so rare that they may be regarded as curiosities.

Injuries to the external organs of the body.—The commonest injuries incidental to athletic exercises are contusions, sprains or strains and fractures. Injuries to the internal organs are happily rarer, for, as a rule, they are of most serious import.

Contusions.—Contusions are met with in every degree, from a mere superficial discoloration in the skin to extensive and deeply-placed collections of blood which may extend far and wide along the facial plains of the body, or become encysted and form large hæmatomata. The principles of treatment are directed towards checking and limiting the amount of extravasation as soon as possible, and as completely as possible. Text-book teaching would have us use cold or hot applications for the purpose. Neither of these are to my mind to be recommended. Cold, to be of real service, must be applied immediately and be intense. The applications of cold are seldom convenient or handy, and they are successful to but a limited degree; further, intense cold is seldom liked by patients, and may even do harm. Sloughing and even gangrene have followed its application to delicate persons the subjects of albuminuria and diabetes. The same may be said of hot applications; to be of real service they must be used at a temperature intolerable to the skin of the average patient, and if of any degree less than this they increase the tendency to hæmorrhage rather than diminish it. A far more helpful remedy is elastic pressure, for it is convenient, easily applied, ready to hand, almost universally applicable, generally efficacious and comfortable. All that is required is some cotton-wadding and an ordinary domette or calico bandage. The wadding, in sufficient thickness, is smoothly laid over the part or limb, and the bandage firmly applied so as to keep up constant compression on the wadding. A rubber bandage is not suitable. The effect is that the pressure of the bandage being higher than the blood-pressure all further oozing ceases, and the fluid portion of the extravasation is readily driven into the lymph-spaces and thence carried indirectly onward into the general circulation through the veins. As the swelling subsides the bandage should be removed and re-applied so as to keep up continuous pressure. In a varying space of time, usually in a few hours, even large collections of blood may be removed by these simple means. The blood that has clotted in the tissues may be broken up and dispersed by gentle friction massage. Massage should not, however, be commenced before at least twenty-four hours have elapsed, otherwise fresh extravasation is likely to be poured out, and the bruising made worse instead of better. To compare experimentally the effects of elastic pressure and the effects of early massage a patient with two black eyes may be selected. If to the one orbit massage is applied and to the other elastic compression, I venture to state that the return to the normal condition will be much quicker in the latter than in the former. Cold and hot applications also suffer by comparison. Massage, though very helpful in the later stages, may be even harmful and delay considerably the cure if used too soon. This is a fact which I believe is not generally realised or accepted by practitioners, for it is the fashion nowadays to employ massage at all times and for most purposes When the collections of blood are large and there are no potent contra-indications, either aspiration or a free incision made with strict aseptic precautions, and with or without subsequent drainage, is very often the most rapid and satisfactory way of dealing with them.

Sprains.—The next in frequency are sprains. When the twisting or wrenching violence is sufficient to overstretch the ligaments of a joint but not to tear them, so that the only symptoms are pain and impaired movement, the condition is known as "strain." When laceration results and there is bleeding into the tissues, and added to the pain and loss of function we have swelling, the term "sprain" is used. Sprains are divided into articular and muscular. Articular sprains may be simple or they may be complicated by fracture or gross injury to nerves, or by semiluxation in the joints. When the sprain is complicated by an injury to the bone entering directly into the joint

it is called a sprain-fracture or fracture-sprain. If in addition to the sprain of a joint there is a fracture of one of the bones in the immediate neighbourhood, but not involving the joint itself, the expression "sprain with fracture" may be used. This classification for clinical purposes is a convenient one, especially when treatment is considered. In the simplest variety known as strain the patient comes complaining of pain and loss of power. The pain may be very intense. The first desideratum of treatment is relief of pain, and nothing succeeds so well as rest to the injured part, and this is best obtained by means of strapping. Various forms of plaster are used for this; the best, I think, are leucoplast and wash-leather plaster. Massage over the plaster will expedite the cure in the later stages. When, however, there is swelling coming on immediately it is due to hæmorrhage, when later by some hours it is due to serous effusion and is known as deferred swelling. The first requirement is that the effusion should be prevented or checked, and when it does exist to assist by every means its re-absorption. Elastic pressure is as useful for these cases as it is in contusions, and should be applied as soon as possible. If the surgeon is fortunate in seeing the case early he may be able to limit the bleeding considerably, and may certainly prevent in a great measure the serous effusion coming on later. Speaking generally, every hour saved before treatment is begun means a saving of days in the total period of treatment. Fixed apparatuses and rigid splints should never be used. Joints are designed for movement, and should never therefore be treated like fractured bones. A joint which is left alone untreated will in the long run do even better than one that is kept in splints for a considerable time. Examples of this are often to be found in the sprains in the lower animals. The system of placing sprained joints and retaining them in splints for considerable periods is to my mind responsible for the fact that thousands of well-to-do and well-educated people, as well as poorer folk, flock daily to the bone-setters and other irresponsible and unregistered practitioners. The bone-setter, who moves everything by rule-of-thumb, would seem to be even more successful by his haphazard methods if we are to judge by his present-day almost universal popularity. Yet both systems, the fixed and the movable, or the orthodox and unorthodox, would seem to be wanting in something. A scientific discrimination is absolutely necessary if success is to commonly follow treatment, for each case must be judged according to its merits. The once prevalent idea that every injured joint required rest is dying hard, just as it is to be hoped that the more modern idea that everything should be moved will find its limitations, for to subject every injury to early massage and movement will be found to be no less injurious. And more particularly is this true when we take sprain-fractures into consideration, and those sprains which are complicated with gross injuries to nerves. Early massage and movements may do much harm by increasing bony callus, and thereby producing stiffness of the joints in cases where there is fracture, and the recent and now almost universal employment of the X-rays has taught us that a great many injuries formerly regarded as simple sprain are in reality sprain-fractures.

Bone-setters.—A few words in reference to bone-setters, electrical masseurs, and other unregistered practitioners. As I stated before, there are thousands of educated people who daily flock to these electrical and other institutions, largely no doubt attracted by the advertisements in the daily papers of the wonderful and almost magical cures effected even after skilled medical attention had failed. Now, it seems to me that our own profession may be to a certain extent to blame for this, for, speaking candidly, I do think that for some years past surgeons have not been paying the attention that is rightly due to these so-called "little things of surgery." The working of the new Workmen's Compensation Act demands that we should, as a profession, be able to give reliable opinions as to prognosis. Are we to allow quacks and others to do this for us? This is no fanciful picture. I have it from fairly good authority that not only is the public looking to these people for treatment, but that even some insurance offices will accept certificates from them as to dates of recovery, etc. It is often only when the cases become more serious, and are likely to come into court, that the surgeon is consulted. I do not for a moment wish to imply that all insurance offices do this, but I have reason to believe that some do.

Surely something should be done to put an end to this sort of thing, for it is an opprobrium

unto ourselves. And although these remarks are addressed more in reference to cases of gross injury to the bones, muscles and joints, they are not altogether out of place in relation to heart-sprain. I have known young men almost frightened out of their wits by being told that their hearts were enormously dilated, that they must cease to take any exercise and to regard themselves as invalids. Some have even had the dimensions of their hearts mapped out in lines of brilliant colours and have brought home charts of the same, and this has not always been the result of their consulting unregistered practitioners.

The plan of treatment which I adopt, after having made as complete a diagnosis as possible in all doubtful cases with the aid of the X-rays, or an anæsthetic, or both, is to aim at getting rid of the effusion by means of elastic pressure as quickly as possible; then, as soon as the swelling is steadily subsiding or is nearly gone, and not until then, to employ massage. Gentle voluntary movements should also be encouraged from the very first, for they alone will prevent muscle atrophy. The normal voluntary impulses sent down from the brain to an injured part is a best and surest means of promoting active healing and preventing atrophy. Passive movements are of limited utility; they act mechanically and are inferior to voluntary movement, and do not prevent atrophy of muscles. Friction massage is made use of in the later stages and continued until the cure is complete. Every sprain treated by these rational methods should be cured in three weeks. I can safely say that I have never had a simple sprain that was not completely cured so that the individual could perform his athletic exercises as well as usual within three weeks, and I have treated a great number. Sprain-fractures take a little longer; it is quite unnecessary to put even these into splints. They are treated in much the same way as simple sprains, with the exception that massage and active movements should be withheld for at least a week from the time of the accident. Gentle movements, on the other hand, are useful. Sprains with gross nerve lesions should on no account be rubbed. Absolute rest must be insisted upon until the pain has quite disappeared, and movements should be very carefully made when first they are adopted. If the pain returns or is increased the movements should be discontinued. These are the general principles of treatment, and I have been forced to omit many of the details. *Muscular* sprains, like simple articular sprains, are best treated by strapping; it is vain to expect muscle-fibres to unite accurately, for when torn they contract and retract. Fortunately new muscle-fibres develop in time to take the place of the damaged ones. Muscles which are torn across become united by fibrous union. If the tear is a slight one strapping may be applied after the manner shown in the photographs which I now send round; such a plan is all that is needed. The strapping gives the necessary support sufficient to prevent further tearing. Instead of keeping the patient at rest in bed after the limb has been strapped I encourage him to walk about as usual; he must, however, discontinue all violent exercises until the union has had time to become firm. Examples of torn muscles are afforded and produced by various athletic exercises. The hamstrings in broad-jumping, the adductors in riding, the abdominales in rowing are familiar types. The prognosis is, generally speaking, good, for other fibres develop to take the place of the old ones, and other muscles develop in due time to take on the work of the damaged ones, and this to a more or less complete extent. When important tendons are extensively torn or displaced it is sometimes best to perform an open operation so as to ensure accurate co-aptation. Ricked muscles are much improved by rubbing.

Many circumstances predispose to athletic injuries:

(1) A want of proper and careful training is, perhaps, the chief cause. Examples of this may be readily found. During the October term the number of sprains are more frequent than at any other time of the year. This is owing to the fact that the athletes begin vigorous games of football, polo, and hockey without having had any gradual preparation during the vacation.

Most of us are familiar with the strain felt in the arm by the effort of throwing a cricket ball early in the season.

Beginners at rowing are peculiarly liable to injure their abdominal muscles.

Perhaps no muscles in the body require more gradual preparation for sustained effort than the heart and diaphragm, in spite of their ceaseless activities.

(2) The kind of game is important, for while

Rugby fooball accounts for the greater number of injuries, the Association, speaking generally, is responsible for the most serious, for while sprains, wrenches, etc., are more common with the former, injuries to the viscera are often associated with the latter.

(3) Temperature has also an influence. Rupture of muscle is more common in dry frosty weather than in damp and muggy weather.

The teaching of physiology accentuates the fact that cold retards muscular contraction and renders muscle tissue less elastic.

Training.—As a precaution against all athletic injuries carefully regulated and gradual training is essential, and this is as necessary for the muscles, tendons and ligaments as it is for the heart. Men who train carefully seldom suffer even after the most strenuous competition. Physiologists are still uncertain as to the explanation of some of the phenomena observed after strenuous competitions. Recently Dr. Leonard Hill has been investigating the subject. He considers that many of the symptoms are due to want of oxygen in the blood; this gas is partly used up by the tissues and partly displaced by the rapid generation of carbonic acid which takes place after violent exercise. He experimented by giving oxygen to men and animals. Oxygen, when inhaled by an untrained man, enabled him to do as well as when he had carefully trained. When given to old and decrepit horses it improved their stamina; to horses in good condition it had little effect. The symptoms of a man who has "run himself out" are very similar to those in one suffering from mountain sickness. This latter condition is known to be caused by the diminution of the oxygen tension in the air, and therefore presumably in the blood. Panting, nausea, vomiting, headache, vertigo, and giddiness are the symptoms usually experienced. The following are some of the symptoms of a man who has run a hard quarter-mile race, and has, so to speak, "run himself out." At first he feels all right; after a few moments he becomes heavy, his temples throb with occasional shooting pains, he sits with his head drooping between his legs; whenever he looks up he readily becomes giddy. After a few minutes he feels better and rises to go to his bath or to dress, when the giddiness once more returns. Occasionally he complains of a giddy attack, and each time that he gathers himself together to put on his garments, all the time he suffers from violent headache, and until he becomes sick he does not feel really relieved. Occasionally he complains of difficulty in breathing, and feels as if the diaphragm was drawn into the thorax as in a violent expiratory effort. It is probable that these symptoms are those of cerebral anæmia, and that the sickness marks a reaction just as it does after concussion or shock. The modern system, or that in vogue in Oxford at the present time for the training of a sprinter, is somewhat as follows: Training begins eight weeks before the race; work at first is very light, for legs unaccustomed to the track are apt to become stiff if the work is serious at first. No fast work is permitted, and starts are forbidden till after the first fortnight, as the muscles are apt to tear across or break from their bony attachments. After a month or six weeks the work is more severe, longer distances are run to gain stamina and wind, then the distance is shortened down.

Schedule for fifth week of training.—Monday, fast 300 yards; trot a lap with a few (three or four) starts. Tuesday, a walk of five miles or two slow laps on a track. Wednesday, fast lap, following a slow lap. Thursday, fast 220 yards, perhaps also 300 stridden through at three-quarter pace. Friday, walk, or slow work on track. Saturday, either medium lap, then a rest, followed by another medium lap. Starts 110 yards, stridden through at three-quarter pace, or a fast lap of 550 yards. Sunday, a ten-mile walk; easy walking on grass if possible.

It is always a good thing to walk a mile to and from the track. Work should become easier before the race, and the week before should be a slack week; only trial races should be done at least a week before the final race; the athlete should go to bed as soon after ten as possible.

To avoid getting stale.—(1) Work should be easy, a hard day being preceded and followed by a slack day.

(2) By variation of work, for instance running over or under the distance that is to be run on the day of the race.

(3) Going early to bed.

(4) On the slightest suspicion of staleness knock off athletics altogether for a few days and seek a change of scene. It is far better to run untrained than to run over-trained or stale, for in the latter

state the individual is unable to make any further calls upon his nervous energy.

In the hundred yards race the greatest dangers are straining or tearing the muscles and becoming stale. At first the work seems ridiculously light; starts should never be allowed for at least a fortnight from the beginning of training, and especially when the weather is cold.

The diet for a sprinter is very much the same as the ordinary one; a restricted one is apt to upset the digestion. The following menu has been found serviceable:

For breakfast: Fish, eggs, toast and butter, weak tea, or hot water and milk.

For lunch: Cold chicken and salad, or cold beef or mutton, followed by stewed fruit and a milk pudding.

For tea: Only a plain cake and not more than two cups of tea.

For dinner: Fish, joint, a variety of vegetables and toast, stewed fruit and some form of milk pudding, followed by dessert or fresh fruit; wine may be allowed in moderation. Champagne is sometimes allowed to promote appetite and prevent staleness. Many athletes drink plain water or Apolinaris or other natural mineral waters. No smoking is allowed.

Over-training.—Over-training is the result of over-work. The heart feels the strain and the nervous energy flags, the digestion and emunctories become slow, and exhaustion rapidly supervenes whenever muscular or intellectual efforts are made. The term known as "getting stale" is often the result of over-training. The individual suffers from loss of mental vigour, often caused by monotony of work and monotony of feeding. The victim considers everything a trouble, and instead of feeling supremely happy as is the case with persons who have trained carefully, he feels himself good for nothing. The treatment of staleness consists in change in every possible way—change of scene, of climate, occupation and society, as well as of food. Men with poor digestions are very liable to getting stale. Another form of staleness arises from the dyspepsia incidental to over-eating; especially is this to be observed amongst inexperienced rowing men, for amongst this class of athletes traditional systems of training hold sway, and enormous meals, largely of meat, are frequently consumed with the idea that rich albuminous food is essential for muscular energy. When the crew of an eight are being trained together and dieted on the same plan it happens that what one man consumes and thrives upon may be more than sufficient to upset the digestive powers of his colleague. The system also of continually dining together and practically living always together becomes very monotonous, and is apt to produce staleness in some of the members of such a crew. Many men become irritable or morose, and even unhappy as the result of this system of traditional training. I can conceive of no plan more likely to promote staleness in an athlete, and it is a little surprising that these traditional methods should be still believed in.

Dr. COLLIER (Oxford) said that his over twenty years' experience in connection with University athletes at Oxford had taught him that whatever athletic competition an Oxford man went in for it would not be marred by a tragic termination. When a man was on the wrong side of sixty, or even of fifty-five, it was not unusual for him to drop in making an extraordinary muscular effort, and in that dropping to die. At Oxford when an athlete made a supreme effort he occasionally dropped, but he never died. For the last twenty-four years there had been only two deaths on the field at Oxford. One was the case of a football player who had been told he had a very serious lesion of the heart-valve, and had been warned against taking athletic exercises. He disregarded that warning, and he paid the penalty. The other case was in a running man who was not so much a runner as a jumper, and he had taken a preliminary run to warm himself, not exerting himself very strongly, and he was seen to falter, to fall and die. He regretted that in neither case did the Coroner order an inquest as he might have done. He had only come across one case in which the patient showed any sign of acute valvular lesion such as rupture of a valve. Therefore when he had to advise an athlete to give up his athletic work he was accustomed to tell him that if he disregarded his advice he was not likely to come to immediate grief, but he had to look to the future, and ask himself what would be his probable condition twenty years hence. One often saw marked cases of dilatation of the right side of the heart. His attention was called to the question of dilatation of the right side of the heart very many years ago in the explanation given of a case by Sir

William Broadbent. An Etonian had come up as a freshman, and was looked upon as a crack mile runner at Eton. When he started practising at Oxford he was making seconds worse time than at Eton, and he consulted a doctor, who brought the man to him. Dr. Collier examined him in the vertical and in the horizontal position, but nothing abnormal was found. But when he was made to run round the room two or three times quickly he noticed that his epigastric pulsation was much more marked and continuous than one would have expected it to be. More particularly, he developed a bruit, running up the left side of the sternum. At that time he was not familiar with this bruit, and he did not find any account of it in the books. He sent the patient to Sir William Broadbent, and he would read that physician's letter, which said that the murmur was produced in the pulmonary artery, the conus arteriosus, and was a result of a temporary dilatation of the right ventricle and conus. It was one with which he (Sir William) was familiar. The conus was in contact with the chest wall, instead of having a layer of lung between, and during systole it was slightly flattened against the wall of the chest, and that led to an eddy in the current and hence the murmur. He wished to relate a striking example of the kind, that of one of the most famous three-mile runners who had been at Oxford. As a freshman he ran in the University sports and did the record time for the three miles. In the second year he beat his first year's record. He was looking after the man, and the third year he found that the man had developed the murmur in question. It was so slight and the man was running so easily that he did not like to stop him. In the Inter-University three-mile race of that year he ran clean away from all his friends and was running for time, and when he got into the last lap he faltered and trotted in, and afterwards was helped to the pavilion, where for nearly three-quarters of an hour he lay in an almost unconscious condition. The next year he was anxious to run again, but he developed the murmur in Dr. Collier's room, and he sent the patient to Sir William Broadbent, who agreed with Dr. Collier that he ought not to race; he was therefore only allowed to make the pace in the mile race. He only ran two laps. Some years afterwards, at the time of the Boer War, the same man consulted Dr. Collier because he wanted to go out to South Africa as a Yeoman. He passed him, as he concluded he would not have to run. But it happened that on one occasion his company was surrounded by Boers and were having a warm time. A friend of the man was wounded, and Dr. Collier's patient put his friend on his own horse and himself ran for his quarters, which he reached in safety, out-running the Boers. There another old Oxford graduate examined him, and apparently he was then again in very much the same condition as after his last race. A fortnight ago a medical colleague brought to him a rowing man, stating that he did not want him to ask the man or the doctor any questions, but simply to give an opinion as to whether he was fit to row. Dr. Collier examined him stationary, and in a vertical and in a horizontal position, without detecting anything abnormal, and he would have passed him as a first-class life for life insurance, until he made the man run round the room two or three times, and then the epigastric pulsation became evident, and with it the familiar systolic murmur along the edge of the sternum. He gave his opinion that he was not fit to row in a hard race, and that if he did so the chances were that in the last quarter of a mile he would be merely a passenger. The doctor then said the man was sent to him because the remainder of the crew were afraid of him as he turned such a bad colour and became so breathless after a sharp row. He did not think that murmur could be elicited until the man was made to exert himself. He looked upon the prognosis in such cases as very favourable. As long as a man would cut off his strenuous competitions and go in for golf, tennis, sculling, boating, and bicycle riding, and otherwise lead a healthy life, he did not think he was likely to be much troubled afterwards. With regard to the left side of the heart, he was accustomed to advise his athletic friends to make their athletic career a short one, because in the case of a schoolboy who went in very much for athletics of a strenuous kind, there would throughout his school career be a definite amount of hypertrophy of the left side of the heart, and if he continued his athletics for many years there was a chance of developing some dilatation of the aorta, and perhaps, in course of time, aortic regurgitation. For that reason he advised men not to let their athletic career be a very long one. He desired, in conclusion, to call attention to

what he regarded as the over-exaggeration of the dangers of rowing indulged in by some responsible medical men. Recently a book in many ways excellent had been written by a doctor on physical efficiency, and he hoped it would be widely read. There was a preface by Sir Lauder Brunton, and a fore-word by Sir James Crichton Browne, but both those gentlemen disclaimed responsibility for any statements in the book. Speaking of rowing, the author said: "No one should handle an oar who is not sure of the state of his heart and blood-vessels, and no one should enter for training unless he has been first medically examined." That was excellent advice. The author went on to say, "Further, everyone, however strong, should, whilst training, be examined medically every third day." Dr. Collier maintained that, if rowing was such a dangerous pastime as to necessitate a medical examination every third day, the pastime should be given up altogether. The author proceeded: "One of the first of the serious conditions induced is kidney trouble, manifested by albumen in the urine. Not a few of the young men at our universities lose their lives or permanently injure their health by rowing when physically unfit." And he went on to say, "By-and-bye sleeplessness supervenes, and owing to a feeling of unfitness a doctor is consulted, who may, and in all probability will, find kidney trouble present, to be followed by alarming and fatal results. Several sad cases have come to the writer's knowledge." It was a great pity, Dr. Collier thought, that the writer did not come forward to one of the Medical Societies and discuss those cases in full, because he very much doubted the correctness of that assertion. Two years ago he (Dr. Collier) read before the Medical Society a paper on albuminuria in athletes, in which he pointed out that he had recently examined the urines of all the University eight after rowing a trial, and in every case there was a considerable trace of albumen present; in 50 per cent. of the cases there was a very marked deposit of albumen. He also said he had examined many of the University crews at Oxford, and in more than one instance every member of the boat had albumen present. In 150 cases examined there were at least 57 per cent. of adults with definite traces of albumen. He doubted whether any man could run a mile at top speed or row over a four-mile course without showing a considerable amount of albumen in his urine, and he did not think it was likely to terminate in any bad result. In conclusion, he might say that he had been watching athletics at Oxford for at least twenty-four years, and the impression left on his mind was that the benefits obtained, so far as health was concerned, far outweighed any of the dangers which might be incurred, and those dangers could, with reasonable precautions, be easily avoided.

Sir LAUDER BRUNTON said the subject of athletics was one of the most important that could occupy the attention of the Society, for upon its use or abuse depended to a great extent the physique of the country. Like every other agent which had a powerful action upon the organism, athletics could do much harm or much good, according to the way in which they were used. In order to understand their proper use it might be well to take a glance at the physiological effects of exercise. Moderate exercise quickened the heart and respiration, dilated the vessels, quickened the circulation, increased tissue change, warmed the body, and produced a feeling of pleasure and exhilaration. Its effects, when repeated from day to day, were to strengthen the muscles and increase the functional power of the heart and respiration, and render the man capable of greater and longer continued exertion. When the exertion was continued for too long a time the feeling of pleasure passed off and was succeeded by lassitude, fatigue, and even by pain, as when muscles were over-strained day after day, instead of becoming stronger they became weaker, and might become atrophied to a great extent. When any exertion, even though when not continued for any length of time, was more than the organism was capable of meeting, the pulse became very rapid, the respiration very quick and also laboured, expiration being greatly prolonged, a feeling of constriction amounting to severe pain was felt in the chest, the face became blanched and bluish, the movements feeble and unsteady, and not unfrequently sickness came on. For a long time the quickened respiration and pulse which followed any active exertion were looked upon as being chiefly of mechanical origin, but it was shown by Mosso at the International Congress in Berlin that that was not the case, and they were actually caused by a toxin produced in

the muscles during their exertion. He narcotised a dog with opium and found that the injection of the blood of another dog which had been kept at rest before the blood was removed from it had no effect upon the sleeping animal, but if the muscles of the second animal had been tetanised before the blood was withdrawn, the blood injected into the sleeping animal produced acceleration of the heart and respiration exactly as if it had been running itself. That toxin, like many other toxins produced by bacterial growth, or like the venom of serpents, had a power of producing fatigue antitoxins just like bacterial toxins, or just as serpent venom produced antivenins; those toxins had the power of not only removing the effect of fatigue toxins, but of enabling the muscles to contract for a very much longer time than the normal. That effect upon the muscles was almost exactly like that produced by training. The fatigue antitoxins were prepared like other antitoxins or antivenins by small quantities being injected into a horse, and the dose gradually increased until very large doses could be resisted. The serum thus contained fatigue antitoxins. The process of training probably consisted in the body being able to produce fatigue antitoxins in larger quantity and more rapidly than before. After a course of training the muscles were capable of a more powerful contraction and a longer endurance, and exertions which would have brought on symptoms of suffocation and collapse could be undertaken not only without inconvenience but with pleasure. Several years ago he had an experience of that sort. He went to Saas Fé, and the day after he arrived there a friend of his who had already been there for a fortnight asked him (Sir Lauder Brunton) to make an ascent with him. He (Sir Lauder Brunton) accordingly accompanied him next day, but before he had got halfway across the level glacier which led to the ascent his heart was beating as if it would burst his chest. He staggered in his gait and his vision became dim, he nearly fell, and was obliged to return. That that was only due to want of training was shown that a fortnight later he not only made an ascent of the Alp Hügel with perfect ease and without discomfort, but they went on as far as Zermatt, the exertion on his second expedition being, he should think, at least ten times greater than that of the first. If they examined the physical conditions which underlay such a state of dyspnœa and prostration as that which he suffered on his first attempt they found that the right heart had become exceedingly dilated, so that the apex-beat was felt under the sternum instead of in its proper position, and the extraordinarily long-drawn-out expiration was almost certainly due to a tense condition of the capillaries surrounding the pulmonary vesicles and holding their walls apart so that the air could enter out of the lungs only slowly and with great difficulty. The fulness of the right side of the right ventricle led, of course, to great venous congestion, and perhaps that congestion affecting the stomach was the cause of the sickness which came on. That, however, was a question into which he need not enter. One would expect it to produce renal congestion and albuminuria, and as a matter of fact albuminuria certainly did occur after exertion in a very much larger proportion of cases than one would at first imagine. The cause of such albuminuria was not definitely ascertained, and it might appear when the exertion did not cause dyspnœa. As a rule that passed off, but if continued for too long a time one would expect the kidneys to become damaged. Increased size of the heart after exertion could not only be made out by alteration in the apex-beat but by percussion, but it had been shown also by the Röntgen rays. If violent exertion were taken only once, the heart, if healthy, soon recovered its tone, but if feeble it might remain dilated for days, and if the strain be frequently repeated the dilatation might become permanent. It was very difficult indeed to say how far that increased size of the heart was due to dilatation only of the right ventricle and how far the left also participated. In such cases he thought it was, sometimes at least, the right side only that became affected, for in cases of cardiac enlargement following strain and accompanied by great shortness of breath in growing boys he had detected no mitral murmur. On the other hand, he had known a mitral murmur come on in a girl every time she went to a dance, and it persisted for two or three days afterwards. Very frequently indeed also one was able to bring out a mitral murmur in cases of feeble heart by merely asking the patient to raise a weight, and many cases also occurred where a mitral murmur came on simply from the excitement of a consultation, although it was not present in ordinary circumstances. The danger of gymnastic exercises without training was brought prominently before him a few months ago by a doctor who came with his daughter to consult him. She had been perfectly well and strong, but had been appointed games' mistress at the school where she was teaching. The strain proved too much for her heart, and her father was shocked to find on auscultation that a mitral murmur had developed within three months after taking up her new duties.

That case illustrated what he thought was now fairly generally accepted—that athletic exercise which is too great for the individual might bring about serious cardiac mischief. The effect of over-strain upon the lungs was usually manifested by hæmorrhage. In the experience he had at St. Bartholomew's Hospital in the Casualty Department, where he usually saw from seven to eight hundred patients a week, he observed that hæmorrhage from heavy lifts and strain of that kind

usually did not come on at the time but about three days afterwards. He did not know how far hæmorrhage from strain would come on in perfectly healthy lungs. It was a matter of common observation that hæmorrhage apparently in healthy lungs might be the immediate forerunner of phthisis, but he thought it was very likely indeed that the tubercle bacillus might have previously found a nidus in the lung and have been the cause of the hæmorrhage. They did not yet know how far albuminuria caused by over-exertion might ultimately lead to kidney damage, but he did not think that that was likely to be of much importance because the organ that was most likely to suffer first was the heart, and alterations in it would probably prevent the indulgence of athletics to any great extent before the kidney itself had suffered. It was, however, clear that athletics without previous training, or with imperfect training, might be very injurious, and that over-strain, especially in growing boys, might lead to cardiac weakness, which might ruin their future lives. It was therefore very advisable that all those who took part in athletics, whatever their age or their sex might be, should be subjected to careful medical examination, unless they were willing to take the risk of doing themselves great damage. Such examination was all the more important if athletics, gymnastics and physical training was introduced as part of the curriculum in all schools.

Dr. SAMUEL WEST said he thought the indication as to the use of massage in damaged joints was pain; if there were pain which lasted after the massage was finished, then the massage in such a case did harm. If, however, the pain was caused merely by the stretching and straining of the tissues and did not last when this was over, there was not harm in the procedure.

With regard to what had been called pulmonary or physiological emphysema, his view was that strictly it was not emphysema but a hypertrophy of the lung. The lungs hypertrophied when they were used, just as the muscles did. The lungs also became larger, and therefore there were produced the physical signs of enlarged lung, which were the same as those of common emphysema, but due to a transient cause. It passed off when training ceased. It was akin to hypertrophy, and not in any way to atrophy, which was the prime factor in ordinary emphysema.

With regard to the effect of athletics upon the heart, when the heart was exercised in training it was agreed that it hypertrophied. Hippocrates said all athletes were in a critical state of health and liable to break down, and it was recognised that over-training was very common. When the heart hypertrophied it did so to some extent at the expense of its reserves, for the normal heart had considerable reserves. The problem of training was how to develop the heart to the maximum so far as hypertrophy went, without diminishing the amount of reserve which that heart had, so that it could produce the maximum amount of effect in a given time. The different kind of training necessary for a long course as compared with a short one was an illustration in point. A person might be able to produce a larger amount of work for a short space of time without damaging his heart than he could for a long time. It was a matter of delicate adjustment, and it was in that that the skilled trainer excelled. When eight people in a crew had to be dealt with, so as to bring each man in that crew up to his best efficiency for that race, the matter was a still more difficult one.

Just as the muscles in general and of the heart in particular hypertrophied, so did the muscles of the vessels, and it was not uncommon to see young people who went in for athletics with thickened vessels, so that, if other things were equal, they would have been thought to be suffering from ordinary arterial sclerosis. The three causes of thickened arteries were: atheroma in old age, granular kidney, and the hypertrophy in athletes.

With regard to the albumen present in the urine of such young athletes, he did not agree that it was in any way dependent upon seminal discharges or evil habits, such as masturbation, though albuminuria could be produced in that way. He looked upon it as an effort to get rid of some of the incompletely decomposed, albuminoid substances which had been produced by the fatigue of muscle; it was a transient condition and had no permanent effects.

The last point he would refer to was suggested by the models of hearts which had been passed round by Sir Lauder Brunton. It was always dangerous to venture to differ from physiologists of eminence, but his objection was to the dogmatic statement which was often made, that muscular action was necessary to enable the valves to close the apertures of the heart. That statement, he believed, rested originally on the experiments of Ludwig and Hasse, and they had been copied from one book to another, and, so far as he knew, had never been repeated and confirmed. The experiments in question were very violent ones. He thought there were many clinical reasons, which he could not mention then, why the heart should not be considered so delicate an organ that any little structural defect could cause gross valvular deficiency.

That point raised another, namely, the cause of dilatation murmurs. If the murmurs which had been mentioned as due to dilatation were due to regurgitation through the valves, why were not they propagated in the direction in which those murmurs were when they were due to organic disease? The organic murmurs were not propagated in the same direction as those under discussion as a rule. The

majority of them, he thought, were not due to regurgitation through the orifices but to intra-ventricular conditions.

The Society ought to be much indebted to those who had brought forward and sustained the discussion, for the subject was a very important one and raised many difficult questions upon which every one ought to have an opinion. Every speaker seems to have arrived at the same general conclusion, namely, that given a healthy individual even violent athletics might be indulged in, and that, too, even with temporary disadvantage, without any serious result following, and that the great benefit of athletics in general outweighed any possible risk of injury.

Dr. DE HAVILLAND HALL said his experience of cases of injury or ill-health following athletics had been almost confined to heart trouble. He remembered three illustrative cases. One was that of a young lady, æt. 15 years, who cycled in Wales, and her ambition was to do some steep ascents. After she reached a good height she got off her machine and collapsed, and as a result she was on her back for six months. She had not had any previous training, and it was evidently a case of over-strain. Even at the end of six months there was great dilatation of the heart in the left ventricle, as shown by the displacement of the apex-beat and the increase in the area of cardiac dulness. Eventually, by means of rest, followed by gradually increasing exercises, and the administration of digitalis, strychnine and arsenic, she made a good recovery. Shortly afterwards he was consulted by a young lady, æt. 19 years, who had been riding her horse and it had bolted with her. As the result of the strain she was very faint and had to be almost carried home. It took at least a year before she got back to her normal condition. After even slight exertion she got a murmur in the mitral area, and therefore he could not say that her heart had yet regained its normal condition. In young people, under suitable treatment and rest, their recuperative power was so great that usually the heart settled down eventually into a useful organ. The third case was that of a boy, æt. 12 years, who was at a preparatory school and was champion of the football team and of the cricket team, and went in a good deal for sports. In the summer time he was going in for the high jump, and immediately after executing a good jump he complained of discomfort in the cardiac region, but he did not make much of it because he wanted to continue his jumping. He went in for some more athletics, and shortly afterwards he got pale and short of breath, and when he, Dr. Hall, saw him he had well-marked dilatation of the left ventricle with mitral regurgitation and tachycardia, and from being a healthy, brisk boy he became rather anæmic and listless. He was now gradually improving under treatment, but he, Dr. Hall, had felt obliged to prohibit all football for him during the present season. Such cases occurred in young people who presumably had had no proper training, and as Sir Lauder Brunton pointed out, now that athletics were to be a definite part of the education in elementary schools, all children, especially boys, before going in for athletics should be carefully examined. Much harm had in the past accrued to children, especially at preparatory schools, through their being allowed to go in for sports when their condition was not fit for it.

Dr. VOELCKER said he thought Dr. Havilland Hall had mentioned what was a very important point in the discussion, namely the distinction between properly regulated athletic exercises and the violent strains to which a good deal of reference had been made, such as in the case of champions at twelve years of age. In the latter aspect there was the key to some of the mischief arising from the abuse of athletic exercises. He could claim to have had a good deal of athletic experience in his younger days, as he had engaged in a fair number of races and was familiar with the conditions, and some of the races had been for fairly long distances, and he believed he was not now a bit the worse for it. He had come across very few cases among his friends, who included a good many athletic acquaintances, who had developed even imaginary mischief with their hearts. He thought it was necessary to distinguish, in regard to their gravity, between different forms of athletics. He regretted Dr. Collier had had to leave, as he had wanted to ask him about rowing. From what he had heard and seen, it seemed to him that rowing men were much more liable to heart mischief than men engaged in other forms of athletics. He believed there were on record more cases of sudden death in men who had been rowing men than in those who had been running men. Of course the effect of simultaneous arm, leg, and breathing exercises was likely to be more severe in rowing than in running. In regard to boxing, most medical men had come across cases in which definite valvular disease, possibly rupture, of the valves was attributable to direct trauma. He wished to join issue with Dr. West in regard to the use of the term "emphysema." Dr. Voelcker thought Dr. West had suggested that it should be spoken of as a hypertrophy of the lung, but Dr. Voelcker suggested an improvement on that by saying it was a distension of the lung and not a hypertrophy. It was one of the dangers associated with the immoderate use of exercises. He had been struck by the number of young men, especially in the services, who had that condition of "emphysema." He did not regard it as a degenerative change, or as a hypertrophic change; he thought it was a mechanical distension of the air-vesicles, and if

the cause of it were repeated it became permanent, and after forty-five years of age would show itself in the same way as one found emphysematous conditions showing themselves in the general circulation and the liability to bronchitis, with subsequent changes in the lung. He thought one of the reasons which made it a real danger was the unfortunate competition to see which form of exercise enlarged the chest most. He was sure those who had seen many athletes would have been struck by the fact that the athlete's chest was not the athlete's chest as depicted by the artist, nor was it the chest of the advertisers who promised to secure so much increase in the chest measurement as the result of their particular exercises. With regard to Dr. Whitelocke's reference to temperature and its effect in producing injuries, he (Dr. Voelcker) thought the greater number of accidents in the winter was due to the frozen and hard condition of the cinder track in the winter, which produced more jar to the body in running, rather than anything else. He did not believe cold increased the friability or lacerability of muscles. He had hoped to have heard some explanation of the phenomenon known as "the stitch," a condition of things which made many men unreliable in a race. He had never come across a proper explanation of it, and he would like to hear what view Dr. Whitelocke had on the matter.

Mr. Lockwood said he would only briefly refer to a fact which had not been mentioned by any of the speakers. A few years ago a sailor was brought under his notice who had thrown a cricket-ball and in doing so had broken his right humerus just below the deltoid, causing a rather complicated fracture. One of the fragments of it was stuck into the musculo-spiral nerve, so that it became necessary to free the nerve and unite the fracture. He was rather puzzled to know why the arm should have been broken merely in throwing a cricket-ball, but on looking at the X-ray photographs he remarked that the sailor seemed to have a woman's humerus and a man's thigh, and one of the relatives said "they always thought something would happen from those Sandow exercises." Evidently people who went in for those exercises might develop their muscles in a short time, but the development of the bones could not keep pace. He was sure all the surgeons present would endorse what had been said by Mr. Whitelocke and approve of his methods of treatment.

Mr. Whitelocke, in reply, said he had himself seen fracture of the humerus resulting from throwing a cricket-ball in the person of a boy, æt. 15 years, who he did not think had had any experience of Sandow exercises. With regard to massage, his experience had been that massage was very seldom painful unless there was some inflammatory process superadded, and of course if any form of inflammation existed, massage should be strongly contra-indicated. In cases of sprain or even sprain-fracture he found that his experience was that massage rather soothed the majority of cases than caused pain. In the exceptional cases where pain was caused by massage, he agreed with the remarks of Dr. West, that the massage should be desisted from. He disagreed with Dr. Voelcker with regard to the effect of temperature on muscles. He (Mr. Whitelocke) was certain that if a man did not train himself carefully and he ran in the winter, even upon grass, he was very liable to rupture his muscles and tendons. He was surgeon to the athletic club and the football club, and he had had ample opportunities of studying innumerable sprains, and he attributed part of the cause of the cases which occurred in the early autumn to the cold temperature. Men would go about the football field in scant clothing waiting for their opponents to come, and as a sequel he saw many sprains and strains of muscles. At Oxford no important cross-country or sprinting event was entered into unless the competitors had had about twenty-one days' training. The hamstring muscles were particularly liable to be affected. In the summer months such accidents were very seldom met with. Physiologists would agree with him that in cold weather muscle was much more friable and less elastic than in the warmer weather. It was customary for a professional runner to douche his muscles with warm water or rub down carefully before setting out for a big race. He would have liked to have heard expressions of opinion himself concerning the nature and causation of "stitch." He thought it was a spasm, probably in the diaphragm, or in some fibres of the diaphragm. He had also hoped to hear a reference to "second wind." He formerly thought it was due to a correlation between the distension of the air-vesicles in the lung, which some members had called emphysema and others hypertrophy, and that it came on after the lungs had expanded sufficiently to give room in the air-spaces where they had already been overcrowded by blood. But the recent investigations of Haldane in the production of apnœa seemed to show that the relative proportion of the carbonic acid in the air-vesicles was the key to the situation. By breathing very vigorously for a few minutes the blood in the lungs could be made to wash out a certain amount of the carbonic acid in the air-vesicles so as to reduce its quantity, and as soon as that was done it seemed to have the effect of diminishing the stimulation of the inspiratory centre. He had seen Dr. Haldane sit down and inflate his lungs and in this way bring about a complete apnœa lasting for three minutes. He thanked all who had taken part in the discussion.

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A CLINICAL LECTURE ON VENESECTION.

Delivered at Guy's Hospital.

By W. HALE WHITE, M.D., F.R.C.P., Senior Physician to the Hospital.

GENTLEMEN,—It happens that we have recently had to bleed some patients in the wards, and we have discussed whether we should bleed several others. Therefore I thought it might interest you this afternoon if we were to go over some points about venesection. It is an extremely interesting matter in the history of medicine, because if you had been here seventy years ago you would have found that most of the patients in the hospital would have been bled, but now hardly any of them are bled, and a matter in which there has been such a revulsion in practice must be interesting to us all. I have heard Sir Samuel Wilks say when he was a young man so many people came regularly to be bled every spring and fall in the year—the public thought these were the best times to be bled—that you might see them lying in rows in the surgery just recovering from severe bleeding, and when a man felt he had recovered he got up and walked away, and nothing was said about it. Seventy years ago legal proceedings were taken against a doctor who did not bleed a patient who was suffering from acute pneumonia. Indeed, not so very long ago many healthy people were bled, and so important was the treatment considered that most sick people were bled. Now venesection is very much less frequent, and twenty years ago many students might go through their whole curriculum without seeing bleeding carried out. For instance, to show that that was so, Jürgensen says: "What I have seen of it has been incapable of leaving any doubt in my mind that this treatment is seldom, perhaps never, imperative." That

represented the feeling of some twenty or thirty years ago, and I hope to show you that it is wrong. It was in the fifth and sixth decades of the last century that the strong feeling against venesection appeared, and it is waste of time now to read the enormous literature connected with this change of opinion, especially as sometimes writers did not express themselves temperately. But you must not go away with the impression that there was no reason for a change. It really is terrible to think of the number of people who in the past have been bled to death. I am certain that if we had to choose between indiscriminate bleeding as practised seventy years ago and for many years before that, and no bleeding at all, we should be very much better off with no bleeding at all. I think it may interest you historically if I take three cases from different authors showing how much bleeding was done some time ago. I took them quite at random. And the first one I happened to pick up in connection with it was from Abernethy's 'Essay on Injuries of the Head.' Here is the case of a boy who was suffering from a depressed fracture of his skull and a meningeal hæmorrhage; so you see he had already, by the nature of his accident, lost a considerable amount of blood. His age was fourteen years, and he had fallen from a scaffold nearly two stories high and pitched on his head. When he was brought to the hospital he appeared to be in an almost dying state. A depressed fracture was found, he was trephined, and when the bone was taken away a large amount of coagulated blood was discovered. The coagulum was removed. When that had been done, fluid arterial blood gushed out in quantities. Then more bone was removed and more clot was taken away, and the operation was finished. You will admit that that boy must have lost a considerable amount of blood. He was put back to bed, had intermitting pulse, was insensible, very feeble, with laborious interrupted respiration. Now Abernethy was one of the greatest surgeons of the day, undoubtedly, and therefore his treatment was the expression of the best opinion then available, but he says: "I now took twelve ounces of blood from his arm. The third day he was disturbed and less rational, accordingly eight ounces of blood were removed from his arm. And on the sixth day symptoms of irritation again took place and were relieved by similar treatment." And ultimately the boy died. So there is a youth who has lost a quantity of blood from the nature of his accident, but who is, nevertheless, freely bled afterwards. In the light of our present knowledge that seems entirely wrong treatment. Take another author. Here is one of the most interesting books in medicine: it is Marshall Hall's 'Researches Principally Relative to the Morbid and Curative Effects of Loss of Blood.' And it was probably the publication of this book as much as anything which did good in pointing out to the profession how much harm was done by taking away too much blood. Here is one of the cases he quotes with disapproval. It is taken from the 'Lancet.' A muscular man, æt. 57 years, was brought to the hospital because he had fallen from a scaffold, and many ribs were found to be broken. He was at once bled by the dresser to eighteen ounces. At noon he was ordered to lose twenty ounces more. He passed an indifferent night. The following morning he was bled to eighteen ounces more. At noon he still complained of pain, his pulse was full and rather jerking, and he was bled to eighteen ounces more, and a similar quantity in the evening. So in that day he was bled to the extent of eighteen ounces three times, although he was bled twice the day before. Two hours subsequently another surgeon saw the patient and concurred with the surgeon under whom the patient had been, and admitted that more blood should be drawn, and consequently twenty ounces more were taken away. The man died. The post-mortem is interesting. The right lung was healthy, but appeared to be very light coloured from the almost total absence of blood in it. The vessels were quite empty, and so on. So there you see, according to our present lights, that unfortunate man, who suffered from no greater injury than having fractured a few ribs, was literally bled to death, being bled to this enormous amount. To show how widespread the custom was let me read you an American case. The patient was a girl, æt. 20 years, who came into the hospital in 1831. There was nothing very much the matter with her, and her illness seems to have been quite vague. Nevertheless, by the time she was twenty-one she had already been bled ninety-three times for her various vague illnesses—once every ten days for two and a half years. The girl herself was, when her physician saw her, in a nervous state. She wanted him to

open the vein, but he declined. But ultimately her persuasion overcame his scruples, and next day, he said, "Unwillingly I opened the vein for the ninety-fourth time." I need not go further into the case except to say that after some time she died, and no disease of her heart was found, in fact nothing whatever was found the matter with the girl at all. She appears to have suffered from various neuroses, for which she was bled ninety-four times, and ultimately she died.

It is clear that our ancestors before about 1855 did bleed to great excess, and often to such a degree as ultimately to kill the patient. And in those days, owing to want of cleanliness, they sometimes had serious trouble in consequence of performing venesection. For instance, Abernethy has written an essay on the occasional ill-consequences of venesection. Here is a case of a lady who was bled in the median basilic vein, and eight days afterwards there were two swellings on the site of the bleeding and both these had irritated the lymphatics leading from them, and both of them were inflammatory as a result of some dirty knife used at the bleeding. Here is another case of a man, æt. 35 years, who was admitted to St. Bartholomew's Hospital, sent up from the country, for a bad arm after bleeding. The whole arm was greatly swollen, the wound made by the lancet was not united, two large abscesses had formed, etc. So there was always a danger of septic inflammation from bleeding in those days.

You may fairly say to me, what are the reasons for which you should bleed? There is no doubt whatever, I think, that the great majority of patients for whom bleeding is desirable are people in whom the right side of the heart is over-gorged with blood. That is most often a result of disease of the mitral valve, or a result of bronchitis or pneumonia. You recognise the condition by noticing that the patient cannot breathe properly, he is very livid, the tips of the ears and nose and the lips are livid, the finger-nails are blue, and the breathing is very hurried and distressful. The pulse is small, and sometimes by percussion you can make out that the right side of the heart is dilated. And no doubt if such a patient were well enough to be X-rayed you would be able to see that the right side of the heart was distended. To show you the good which can be done for a case like that, let me give you a quotation from Sir Samuel Wilks; it is so graphic. He says: "You see your patient sitting up in bed, the face, tongue and lips blue or purple, and the jugular veins starting out of the neck and often visibly pulsating; the heart beating quickly and perhaps a tricuspid bruit, indicating the gorged right heart and obstructed lungs; the veins in the body are full to bursting." That is the most satisfactory kind of case to bleed. It has been said theoretically that bleeding would not do any good for such a case owing to the valves in the veins. All I know is that it does do good, and the distressing condition immediately passes off. Possibly the veins are so stretched in such a case that the valves do not act. At any rate there is no doubt that if you bleed a patient who conforms to the picture drawn by Sir Samuel Wilks you will see the lividity pass off, the pulse will become better, and the breathing quieter and less laboured. Further, as after bleeding in such a case the blood can flow better through the lungs, oxygenation of it takes place with greater facility.

The next question is, How much should the patient be bled? There is no rule for that. But the average amount is ten to fifteen ounces. In a minute or two we will consider the circumstances which should guide us as to the amount to be removed. Another point is, I am often told that a small pulse in such a case as that we have just considered is a reason why you should not bleed. It is stated by the thoughtless that the smallness of the pulse is an indication that bleeding would be fatal. But the fact is that a small pulse is the very reason you should bleed, the interpretation being that so much blood is dammed back in the venous system that not much of it gets on to the arterial system and the pulse is consequently small. You will often see after bleeding in the kind of patients we are describing that the pulse will improve considerably. Another thing you will notice is that sometimes in a cardiac case if the patient does not respond to digitalis when he is in this livid condition he will respond to it after the bleeding. That is not surprising, because in the first place the circulation is very poor before the bleeding, and in the next place the heart muscle, like any other muscle, is full of venous blood, and digitalis does not get a chance until the patient has been bled, and it certainly cannot act on the right ventricle until

the patient has been bled, because the right ventricle is in a state of paralytic distension; and therefore cannot contract in response to the digitalis. Another way in which bleeding may do good is this: you know it has been shown by Professor Lorrain Smith that in heart disease the total volume of the blood in the body is increased. That seems reasonable. To show how much it may be increased I give you the case in which Professor Lorrain Smith worked out the problem. It was that of a boy with adherent pericardium who was in the Belfast General Hospital. While he was in the hospital he became worse, his circulation failed, and he developed ascites and œdema. The quantities were these: the boy gained 11 lbs. in weight in a given time, and the total volume of his blood increased from 2557 c.c. to 3355 c.c. If other heart cases are like this, you will not be surprised to hear that bleeding may be of very great value, seeing the large amount of blood there is in the body, because the diseased heart has then more blood to cope with than it can manage. As showing the value of loss of blood remember that many people with heart disease have frequent hæmoptysis; it is one of the common symptoms of mitral disease. There was a woman—I have not seen her lately—who frequently came into the hospital for a night or two under my care. She was able to earn her living as a washerwoman. She came in for hæmoptysis, having mitral stenosis, and she was always very much better for the hæmoptysis: it was Nature's bleeding for her. We have had such cases in Miriam Ward recently. There is now at the present moment a woman in No. 25 bed with mitral stenosis. I did not see her on admission, but she was bled because she was livid, and I am told the bleeding did her good, and I am prepared to believe it; I should think without doubt that it did do her good. It is the class of case you should bleed. The other day there was a man in No. 3 bed in John Ward with acute bronchitis. We bled him, his lividity became less, his pulse became better, and he felt better in every way. Later on in his illness the lividity returned, we wanted to bleed him again, but his mother would not allow it, and he died shortly after. I think if we had bled him we should have relieved him very considerably. The most striking cases I will relate are two which I saw not long ago. A vat of nitric acid broke in a cellar in London, and the fumes came up through the grating, and passers-by thought there was a fire and summoned the fire-brigade. And the firemen also thought the fumes of nitric acid were smoke, and they descended into the cellar and inhaled these fumes. Many of the firemen suffered from very severe capillary bronchitis due to this irritant. I was asked to see two who were so bad that it was thought they would die. The first man I saw was a magnificent specimen of humanity, a great big, tall, well-built muscular man, weighing about sixteen stone, sitting up in bed, deeply purple, with a small pulse, rapid, and breathing with the greatest difficulty and distress, sweating profusely. The opinion of the doctor who saw him was that he was dying. He was certainly getting worse very fast. His lungs were full of fine crepitations. We bled him till he felt faint, twenty-three ounces being taken away. He said he felt better after that, that he had not felt so well for two or three days. From that time he never looked back, and he is now perfectly well. The improvement in the colour and in the pulse in that case was most striking. For my own part I have no doubt that the bleeding saved the man's life. The other man, who was not so bad, was also bled, and I think the bleeding in his case contributed to his recovery. I might spend the whole afternoon quoting to you cases of engorged right side of the heart which have benefited from bleeding. The best collection of such is one published by Dr. Sutton, that great genius who was on the staff of the London Hospital. And here is one of them. A man with mitral disease was admitted with breath very short, veins in the head and neck much distended. Next day his face was livid and swollen, the veins still distended, great dyspnœa. He could not get any sleep on account of his breathing. Sixteen ounces of blood were taken from the arm, and he expressed himself as feeling much relieved. The veins in the neck were less distended and his breathing continued easier. He went out of the hospital very much better. Here is another one: admitted with great dyspnœa, a loud crowing systolic murmur over the heart, œdema of feet and legs, veins greatly distended: venesection sixteen ounces; felt much better. The next day still better. A few days later he became bad again; respirations, fifty-six. Bled to twenty ounces and felt much relieved. Here

is the case of a man who was suffering from disease of the mitral valves. He had great dyspnœa, and was bled at once to twelve ounces, and expressed himself greatly relieved. Here is a man who had had rheumatic fever, much albumen, disease of mitral valves, very livid, much shortness of breath, gradually became worse and worse. Venesection to ten ounces, improved considerably. I might read you many of those, but they are all to the same point, namely, that when the right side of the heart is very distended and the patient very livid you may bleed them with very great benefit.

These instances of mitral disease of bronchitis form one group which is benefited by bleeding. The next group that may occasionally be benefited are people suffering from pneumonia. You might think that as the lung is solid in pneumonia there would be great retardation of the blood on the right side of the heart. As a clinical fact that is not often so. Everyone will bear me out that the majority of people with pneumonia are not livid: it is quite rare for a person with pneumonia to die from respiratory failure: they die from cardiac failure. But occasionally you do see a person with pneumonia who is very livid. Such a person will be benefited by bleeding. There was a man a few days ago in No. 14 bed in John Ward. He had pneumonia, he was very livid. We bled him, and he was improved by the bleeding: his lividity was less and his pulse better. We hardly hoped to save his life, for his pneumonia was very severe and he was an alcoholic subject. Nevertheless, there is no doubt the bleeding did relieve him temporarily. Here is a case showing how pneumonia may be relieved by bleeding. It is recorded by Sir Stephen Mackenzie. A lady who was suffering from acute pneumonia was cyanotic, pulse 120, temperature 104° F., respirations 52, stupid, drowsy, apathetic. The doctors drew off twenty-six ounces of blood. Next morning the temperature was 98·2° F. In the evening it was again 104° F. And next evening her condition appeared to be desperate, she being quite unconscious. Twelve ounces of blood were withdrawn and immediate relief followed. The patient got quite well, and the opinion of those who recorded that case is that the bleeding saved the patient's life. So you see there are cases in which it is desirable to bleed in pneumonia. But please remember those cases are very few, because patients with pneumonia do not, as a rule, die from venous obstruction.

In olden times if your patient had any malady which could by any possibility be ascribed to the brain you probably bled him. You remember even if the malady was entirely functional the patient was bled, for I have told you about that unfortunate girl who was bled ninety-four times before she was twenty-one, probably for hysteria. Still, as bleeding for cerebral conditions was formerly so common it behoves us to consider whether there are any maladies of the brain for which it is desirable to bleed. You might say let us bleed when a man has cerebral hæmorrhage, on these grounds—that it reduces his blood-pressure very much. By those means you may say you may help to stop the effusion of blood into the brain. And Gowers does say that in a case of cerebral hæmorrhage if the pulse is very full and bounding very high, and the heart hypertrophied, the patient is better for being bled. I confess I have not bled many patients suffering from cerebral hæmorrhage, for in most cases when a man is losing blood in one place it is not the best treatment to try to stop it by making him lose blood in another. No doubt if you have a hole in a pipe, and you make a hole in it somewhere else, some of the water may not go through the first hole but will go through the second. But that does not seem to be an ideal way of dealing with the difficulty. Nevertheless there are probably some cases of cerebral hæmorrhage in which bleeding will do good, especially those who are livid. Next let us take epilepsy. The patient, owing to the spasm of the respiratory muscles, is extremely livid, the eyes start out of the head, he is frothing at the mouth, there is blood-stained saliva, and this trickles down his face, and it is altogether a ghastly sight. In such a condition bleeding will sometimes act like a charm. For instance, here is a case recorded by Sir Samuel Wilks: "We were informed he had never been out of a fit for four hours. We found him lying in bed with constant convulsive movements, but the most striking condition was the engorgement of his lungs, his labouring heart and increasing lividity of the surface of the body. I requested that he should be bled. The blood poured out in a torrent, the face rapidly became pale, the man opened his eyes and spoke for the first time since the morning, the interval having been a blank to

him." I think you will agree with me that a case like that shows how severe epileptic fits may be, and it illustrates that such a patient will be benefited if you bleed him. Another condition for which bleeding used to be much practised is uræmia. I have bled people for that. The way in which it may act is this: in the first place the person with uræmia often has convulsions. They are epileptiform. The patient becomes livid like a genuine epileptic, so you may relieve him by bleeding. In the next place, the person with uræmia very often has a very high tension pulse, and the bleeding will relieve that. Further, it has been said if you take several ounces of blood away you diminish the amount of uræmic poison in the system. That you do, but it is so quickly replaced that I do not think much good follows it. But certainly there are people suffering from uræmia who are better for bleeding, and especially is bleeding likely to be suitable if there are severe convulsions or a high-tension pulse. The same may be said with regard to puerperal eclampsia. Bleeding has often been resorted to for that, and with very great benefit, the indications being the same as those for uræmia.

In olden days bleeding was practised for fever. I do not suppose anyone now would bleed a patient because he had fever. It has been done with the object of reducing the temperature, but in the first place reduction of temperature following bleeding is a very uncertain event: bleeding is the most uncertain antipyretic we know. And in the next place our modern conception of fever is that it is undesirable to reduce its temperature; for example, antipyretic drugs are not used for that condition, and I think rightly. I have often talked to you about it at the bedside and pointed out that in pneumonia, other things being equal, lowness of temperature means that the patient will do badly, whereas a moderately high temperature means that he will do well. I cannot stop to go over with you again now the reasons for that, but you know that you must not regard pyrexia as necessarily harmful. Therefore bleeding as a means of reducing temperature is unsound and is very likely to do harm to the patient. And even if the bleeding does reduce the temperature the effect passes off in a few hours, and you cannot bleed your patients indefinitely nowadays although it used to be done seventy years ago.

The only other condition for which bleeding is practised that we need now mention is to relieve the pain for thoracic aneurysm. Occasionally you will find that it does so. Why, I do not know. But patients who are suffering agony from thoracic aneurysm will sometimes beg to be bled, and I often say that the most horrible thing we see in medicine is a really bad case of thoracic aneurysm. These patients cannot lie down, they spend the day and the night sitting by the fire in the ward, afraid to move, because the pain is often brought on by movement, and anything, whether it is bleeding or anything else, which affords the slightest relief of the pain will be welcome to them.

The remaining point concerning the clinical aspect of the matter is—when to avoid bleeding. Our forefathers found out, when bleeding was very much more practised, and I think they were right, that the very young and the very old bear it badly. Be very cautious how you bleed children. I do not like doing it. I would sooner put on one or two leeches in the case of a child. Very old people also bear bleeding badly. You must not bleed very debilitated or very weak people; that is obvious. Women bear bleeding better than men. And that is what you would expect, for every woman bleeds once a month and therefore they get habituated to it. You will of course not bleed people who are very anæmic. No one would make such a mistake. You would not bleed anyone whom you knew to be suffering from the hæmorrhagic diathesis, nor would you bleed anyone who was pale, with bad aortic regurgitation. On the other hand, there are many heart cases in which mitral disease is associated with aortic disease, which are very much better for bleeding. I told you just now that the average amount which you would bleed an adult is somewhere about 10 ounces, but I pointed out that 23 ounces were taken from the man who had bronchitis subsequent to nitric acid inhalation. The right thing to do is to open the vein and watch the patient, and make the character of the pulse and the degree of the lividity your guide. If the pulse, which at first improves after your bleeding commences, begins to get feeble later, stop the bleeding. But the amount of blood which people can lose is most variable. You see the same degree of variability in menstruation in women. Marshall Hall says he has known people who could not stand being bled more than four ounces, and he has known other people lose 60 or 70 ounces without minding it. It is always well to have the patient whom you are going to bleed erect. As a matter of fact such patients nearly always are in that posture, because they are suffering from respiratory distress and so are sitting propped up in bed. Never bleed a patient while he is in the

recumbent posture, because you will have but slight indication when he is turning faint from it.

The symptoms due to taking away too much blood we fortunately hardly ever see to-day. The older authors discussed them at great length. Marshall Hall gives a good account of them. He says the immediate effects of excessive bleeding are pallor, syncope, giddiness, unconsciousness, eructations, and sickness, the respirations are slow and deep, pulse slow and weak, the face and general surface pale and bedewed with perspiration. On recovery there may be momentary delirium, but as consciousness returns the patient yawns, sighs, and breathes irregularly. He is often better after he has vomited. In a fatal case the countenance becomes more pale and sunken, consciousness may remain until the last, generally there is some delirium, the breathing becomes stertorous, the pulse becomes extremely feeble. The extremities become colder and colder, there are constant restlessness and jactitation. At length the strength fails, the patient sinks, gasps, and expires. Fortunately we do not nowadays see those results from bleeding. But they are the same as you see when a patient dies from severe loss of blood after surgical accidents, and you can read them in your text-books.

How does the patient regenerate the blood to take the place of that which he loses? Even while the act of bleeding is going on he begins to regenerate. Fluid pours from the tissues into the vessels and for a time the result of that is that as the fluid pours in so quickly before the solids can be passed in from the body and before the corpuscles can be formed by the marrow, that after a bleeding the blood is dilute. For instance, Davy experimented on lambs, and found when they were bled to death the specific gravity of the blood fell from 1024 to 1018. And that meant the fluids were poured into the vessels before the solids. The specific gravity of the blood in oxen which had been bled fell from 1027 to 1021, showing again the dilution of the blood. So very quickly the blood becomes hydræmic. Gradually the loss of red cells is made up, but after a severe bleeding the number is not normal for about two or three weeks. As you would expect, the red cells are normal in number before they are normal in size and shape. The first reds thrown into the circulation after the depletion of blood vary very much in shape and some are nucleated cells. What happens is that the marrow in its anxiety, as it were, to make up blood, pours into the blood several imperfectly formed corpuscles. The white cells are increased, so after a bleeding there is generally some leucocytosis. Dr. Boycott has kindly lent me some lantern slides showing the rate of regeneration of the blood after bleeding in animals, and these I will now show you, and you will see that they agree with what I have already told you.

March 29th, 1909.

GASTRIC HÆMORRHAGE.

A Clinical Lecture delivered at University College Hospital.

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GENTLEMEN,—This lecture is based upon two cases of gastric hæmorrhage which have recently been under my care in the hospital, and both of which illustrate some interesting and important points in connection with this subject. I shall first discuss the cases and then offer some remarks upon the clinical investigation of gastric hæmorrhage.

(1) *The first case* is that of a young man, æt. 22 years, and a printer by trade. His present illness began just before Christmas, 1907, by the vomiting of a little blood. He took to his bed for a week and recovered. At the beginning of February, 1908, he vomited "half-a-gallon" of blood and was confined to his bed for three weeks. He then noticed a swelling in the pit of the stomach, which swelling gradually became larger and caused him pain on breathing.

He was admitted to University College Hospital on March 5th, 1908. On admission he was found to be well nourished but very pale, the blood showing a moderate degree of anæmia of the chlorotic type. The cardiac impulse was displaced upwards into the fourth left intercostal space. The abdomen was slightly distended and bulged a little in the flanks. No superficial veins were visible. The spleen was considerably enlarged, extending about to the umbilicus. The liver was not felt and the upper limit of its dulness was normal. There was shifting dulness in the flanks, indicating the presence of free fluid in the peritoneal cavity. The only past illness that he could remember was pneumonia at the age of eleven years. He took alcohol in moderation. One brother died of Bright's disease, otherwise there was no serious illness in the family. He was discharged on March 23rd in *statu quo*. He was again admitted on April 9th, 1908, vomiting blood. From April 9th to 11th he vomited nine pints thirteen and a half ounces of blood on sixteen occasions, in amounts of from 1½ to 47 ounces. No blood was vomited after April 11th.

On April 12th his temperature went up to 103° F.,

at which level it remained with daily variations till April 15th, when it fell to normal.

His pulse varied from 136 to 154 during this period of fever. Severe diarrhœa commenced on April 13th and lasted till the day of his death, on April 19th. The motions were light coloured, did not contain blood and varied in daily number from 6 to 21. On the day of death venous thrombosis commenced in the small veins about the nose and right foot. There was œdema and blueness of the nose, upper lip and left eyelids, with epistaxis.

The patient complained of severe pain in the right foot which was livid and œdematous, the superficial veins being hard. By 2.15 p.m. the œdema had completely closed the eyes; the patient was delirious and died at 3 p.m.

At the post-mortem examination many small erosions were found in the mucous membrane of the stomach. They penetrated to varying depths and some were slightly stained by altered blood. No ulcer, acute or chronic, was present. The hæmorrhage had evidently resulted from a general oozing from the capillaries in the mucous membrane, and the erosions, which gave the appearance as if in places the superficial part of the mucous membrane had been rubbed off with the finger, were evidently secondary to patchy disorganisation of the mucous membrane by the hæmorrhage. This of course would aggravate the tendency to bleeding. About five feet of the small intestine were quite black and its veins thrombosed. The thrombosis extended up into the superior mesenteric and portal veins, both of which were occluded by blood-clot. The clotting extended into the splenic vein. The clot in the portal vein was beginning to break down in the centre. There was a little free fluid in the abdominal cavity. The liver was markedly fibrotic and coarsely hobnailed, the liver substance being bile-stained and fatty. The spleen was greatly enlarged, the capsule thickened, the Malpighian bodies and the general splenic substance fibrosed. There was a little fibrosis of the pancreas.

There were a few atheromatous patches in the aorta. The lungs showed a few patches of collapse in the lower lobes. The remaining organs were healthy to the naked eye.

The case, then, was one of splenic anæmia with cirrhosis of the liver commonly known as Banti's disease.

Repeated severe hæmorrhage from the gastric mucosa is common in splenic anæmia, and in this case by relieving the portal circulation accounted for the absence of marked symptoms of portal obstruction.

This case illustrates three important points:

(1) The way in which gastric hæmorrhage is produced in a certain class of diseases.

In this class of case there is presumably some poison circulating in the blood which damages the capillary wall and leads to rupture.

(*a*) In this case, as in other forms of *anæmia*, it is difficult to say whether the damage done to the capillary wall is the result of an endothelial poison, or whether it is owing to degenerative changes resulting from the anæmia. Other anæmias in which hæmorrhage is liable to occur are leucocythæmia and pernicious anæmia.

Many other toxic diseases may produce hæmatemesis in the same way:

(*b*) *The hæmorrhagic diseases*, purpura, scurvy, hæmophilia, although in the latter disease it is possible that there is some injury inflicted upon the mucous membrane, which has not been detected post-mortem. In melæna neonatorum hæmatemesis and also hæmorrhages in other parts of the body may be occasionally seen, no ulceration of the alimentary canal being found post-mortem.

(*c*) *The acute specific fevers*, particularly small-pox and yellow fever.

(*d*) *The toxæmia associated with liver disease*, as in acute yellow atrophy, phosphorus poisoning, and cholæmia.

(*e*) Occasionally in *young women apparently healthy or suffering from menstrual disorder* gastric hæmorrhage is seen, without any lesion of the mucous membrane being present to account for it. This used to be called vicarious menstruation, but it is quite certain that the stomach does not act vicariously in this way, and there is no doubt that the hæmorrhage results from some endothelial poison circulating in the blood. Some of these cases may belong to the anæmic group mentioned above. Such cases have also been called gastrostaxis.

When the hæmorrhage produced by a capillary wall poison is very profuse it is liable to be associated with superficial erosions in the mucous membrane as in the case under consideration.

They are due to disorganisation of the mucous membrane in places by the blood, the gastric juice removing the broken-up tissue. These erosions are liable to be overlooked unless one is accustomed to seeing them. They appear to be of no significance, except that it is possible they may lead to further bleeding. Parenchymatous hæmorrhages may also occur, causing ecchymoses.

(2) The second point to which I wish to refer is the association of fever with gastric hæmorrhage.

In this case the fever started the day after the hæmorrhage had stopped. It lasted four days, and the highest point reached was 103° F. It is by no means uncommon in gastric hæmorrhage whatever the cause. The pulse is commonly markedly accelerated.

The height of the fever appears to vary with the quantity of blood lost and the rapidity of the bleeding.

It is probably due to bacterial action upon the blood-clots in the intestine. The diarrhœa in this patient certainly seems to favour this supposition, and from this point of view it was treated by internal antiseptics (salol and bismuth salicylate).

In cases where a large vessel is opened up it may depend upon invasion of the vessel by bacteria.

It has also been supposed to be due to diminished loss of heat from surface cooling or an affection of the supposed heat-regulating centre.

These are mere suppositions and are doubtless incorrect.

Another occasional sequel of gastric hæmorrhage is blindness, to which I will briefly refer.

The patient under consideration was carefully examined, but no evidence of this detected.

The blindness may come on at once, or at the end of one or more weeks.

It may be a very slight attack of amaurosis, or total blindness, or hemianopia. The sight is usually recovered or some slight defect may remain, but permanent blindness of one or both eyes may result. Some of the cases probably have a central origin and others a peripheral one. Various explanations have been advanced for this condition, such as shock of the nerve elements of the retina, absorption of water from the vitreous, causing malnutrition of the fibres, fatty degeneration of the optic nerve and retina, or a fall of blood-pressure whilst the intra-ocular pressure remains unchanged. There may be no changes in the fundus. Pallor of the papilla with narrow blood-vessels may be seen, and optic atrophy and choked disc have been described.

(3) The third point is the occurrence of thrombosis.

Thrombosis as the result of anæmia, whether resulting from loss of blood or as a primary condition, is well known. It is of the marantic type and affects the medium-sized vessels (cerebral sinuses, femoral vein, and so on). Its origin in this case, in the small veins of the nose, intestine, and foot, however, suggests a terminal infection by bacteria.

In support of this view I may mention that I have recently seen a case of splenic anæmia in which death resulted from severe epistaxis.

Just before death venous thrombosis resulting in gangrene of the nose occurred, and it seems more than likely that a terminal infection through the nose was the cause.

A case exactly parallel to the one under consideration occurred in University College Hospital in 1895. The patient was a woman, æt. 43 years. She was admitted under Dr. Sidney Ringer, on January 30th. She had commenced vomiting blood five days before and for some days previous to this had had "indigestion." She continued her hæmatemesis and melæna for five days after admission, a rise of temperature to 102° F. occurring on February 2nd and 3rd. On February 13th there was cramp-like pain in the left leg and the left foot became œdematous. On the day following the toes became purple and the veins of the foot were clotted. The tip of the nose became cold and purple, she had considerable dyspnœa, and died the same day.

At the post-mortem examination a recent acute ulcer was found in the stomach and the scar of an old one. The gangrene of the foot was due to venous thrombosis, which extended up the tibial vein into the femoral. There was thrombosis in the left pulmonary vein, and a clot in a branch of one of the renal veins.

(2) *The second case* which I wish to bring to your notice is that of a servant girl, æt. 30 years. She was admitted to the Hospital on February 19th, 1908. Her illness had commenced on the day of admission, when she suddenly vomited half a pint of blood; she had five attacks of bleeding on the

same day. No previous history of any stomach disease or indigestion could be obtained.

She was well nourished, blanched, and had a pulse of 120. She had three attacks of profuse hæmatemesis during the next two days and passed black motions. She died on the sixth day after admission (February 25th) in a state of collapse. Her temperature, which was 100° F. from the second day after admission, went up to 103·8° F. on the night of her death.

At the post-mortem examination a recent acute ulcer was found near the cardiac end of the stomach. It was the size of half a threepenny piece and its base was crossed by a vein from which blood could be squeezed. There was much altered blood in the intestines.

I will briefly refer to three points in connection with this case:

(1) It illustrates a second manner in which gastric hæmorrhage is brought about, namely, by ulceration of the mucous membrane, the blood-vessels being opened up from without.

Any destruction of the mucosa will cause hæmorrhage in this way, as, for instance, acute or chronic gastric ulcer, cancer, syphilis, and probably tubercle, or injury, whether mechanical or the result of poisoning.

(2) Another point brought out by this case is that sudden hæmorrhage may be the first and only symptom of acute gastric ulcer.

It therefore follows that in any given case of gastric hæmorrhage unassociated with stomach symptoms, it is quite impossible to diagnose whether the hæmorrhage is the result of an acute gastric ulcer or an endothelial poison acting upon the capillary wall, giving rise to a general oozing of blood. Gastric ulcers produced experimentally heal up in a very few weeks (three or four usually) and may leave no sign that they have ever been present.

It is also by no means uncommon to find acute gastric ulcers in the human subject in infective conditions, and such ulcers apparently may give no clinical sign of their presence. It therefore appears that acute gastric ulcer in the human subject may be much more common than it is supposed to be, but that it gives rise to no symptoms unless an accident happens such as the opening up of a blood-vessel, and that it heals as experimental ulcers do in a few weeks.

(3) The third point worthy of note in this case is the presence of fever, but I shall merely mention this in passing as the subject has already been discussed.

In addition to these two causes of gastric hæmorrhage, namely, a blood poison acting upon the capillary wall, or a breach of surface opening up a vessel, there is yet another. This third cause is *dilatation of the vessels leading to rupture.* Probably in most of these cases a small erosion precedes the bleeding, but the primary cause is the dilatation of the vessel. This dilatation may affect veins, capillaries or arteries.

Varicose veins are seen in cirrhosis of the liver, but as a fact they are usually situated at the lower end of the œsophagus, and form one of the channels of anastomosis resulting from portal obstruction. Dilatation of the capillaries occurs in inflammatory congestion (gastritis) or mechanical congestion resulting from venous obstruction, whether this be the result of heart disease, obstruction of the trunk of the portal vein, or hepatic cirrhosis. An enlarged spleen may also give rise to mechanical congestion of the stomach, as does also the prolonged vomiting of sea-sickness or pregnancy, which, however, very rarely lead to hæmatemesis. Rupture of a miliary aneurysm in arterio-sclerosis is a mere pathological curiosity.

In all other cases the blood *enters the stomach from without* and is then vomited up. It may come from the nose, gums, larynx, lungs, or œsophagus, and be swallowed; it may be regurgitated from the duodenum in duodenal ulcer, or it may come through the stomach wall as in ruptured aneurysm.

Clinical Investigation of Hæmatemesis.

(1) The first point to settle is whether blood is really present or not.

The reasons for this are that certain substances may be vomited which resemble blood, such as claret, the juice of certain fruits, and iron, and that blood may be present, although it cannot be detected by inspection: this latter is known as *occult blood.*

The detection of red blood-corpuscles by the microscope is of course diagnostic, but they are not seen in the presence of acid. The guaiacum test for blood is not reliable if done in the ordinary way. The filtered contents, to which a

third, glacial acetic acid, is added, are extracted with ether, and to this extract a few drops of Tr. guaiaci and turpentine are added. A violet blue colour indicates blood. The spectrum of acid hæmatin may be obtained by adding a few drops of acetic acid to the contents, extracting with ether, and examining the ethereal extract with the spectroscope. The vomit may also be tested for hæmin crystals in the usual way, or for the presence of iron by the Prussian-blue test, care being taken to eliminate the presence of iron in the food.

Occult gastric hæmorrhage occurs in the presence or absence of hydrochloric acid if intimate and long-continued admixture of slight bleeding with the stomach contents has existed. It may be detected in the fæces if the patient does not vomit.

Occult gastric hæmorrhage is absent in gastric neuroses, gastric atony, and achylia gastrica. It is present in variable constancy and frequency in cancer, gastric ulcer, and pyloric stenosis with stagnation. If occult gastric hæmorrhage is present, HCl absent, and lactic acid fermentation with stagnation present, the case is usually one of cancer.

(2) Blood being present, the second point to settle is whether the blood comes from the stomach or not. There are two reasons for this: (*a*) Blood may enter the stomach and be subsequently vomited. (*b*) It may arise during the act of vomiting from the œsophagus, trachea, pharynx, or mouth.

If streaks of bright red blood be mixed with much saliva and mucus, the gums should be carefully examined for pyorrhœa or self-inflicted injury. Vomiting of blood in children should always lead to an examination of the naso-pharynx for adenoids. Bleeding from the œsophagus should be suspected if the vomited matter is alkaline, contains undigested food and considerable amounts of saliva and mucus.

Blood from the respiratory passages is bright red, foamy, and alkaline; it is followed by rusty sputum for several days, and no melæna results. The history and physical signs may be enough to settle the diagnosis. A history of cough at the time is misleading, for cough may result from vomiting, or cough may cause vomiting.

(3) It is very difficult to lay down any general rules for diagnosis from the *character of the blood*, because almost every case has exceptions. The colour of the blood of course depends on how long it has remained in the stomach: one hour is a sufficient period of time for blackening of the blood to occur in. Small amounts of blood indicate capillary oozing, and large amounts usually indicate that a large vessel has been opened up, but, as we have seen above, profuse hæmorrhage may occur from the capillaries.

Coffee-ground vomit indicates slow and more or less continuous oozing, and occurs in cancer, gastritis, and mechanical congestion of the stomach.

Clots of blood dark in colour indicate more profuse bleeding, and occur in cancer, gastric ulcer, and cirrhosis of the liver.

Profuse and bright red blood indicates copious hæmorrhage, usually from a large vessel. Such bleeding occurs in gastric and duodenal ulcer, cirrhosis of the liver, very rarely in cancer, in enlargement of the spleen, and the very rare condition of aneurysm.

Hence of the three great causes of hæmatemesis cancer is likely to give rise to coffee-ground vomit or the vomiting of clots, gastric ulcer to the vomiting of clots or profuse bleeding, cirrhosis of the liver to any of the three varieties.

Absence of hæmatemesis and the presence of melæna is said to favour the diagnosis of duodenal ulcer, but a patient may die from hæmorrhage in gastric ulcer and never vomit at all, the blood being passed by rectum. In cancer of the pylorus with stenosis there is, as a rule, no melæna.

Of the capillary wall poisons, in conditions of jaundice and the infective fevers, the blood is more likely to be of the coffee-ground variety or clots. In the purpuras and anæmias it is more likely to be profuse and bright red.

(4) *Relation to vomiting.* Four classes of cases may be recognised:

(*a*) The gastric hæmorrhage may be the immediate cause of the vomiting. Without any warning blood is suddenly brought up. As instances of this condition may be mentioned, acute (rarely chronic) gastric ulcer, cirrhosis of the liver, in which an œsophageal vein ruptures, and the various blood poisons, in which no stomach symptoms are present, but in which the capillary bleeding leads to vomiting as in the purpuras, anæmias, and infections.

(*b*) During the course of the disease, in which vomiting is a prominent symptom, blood is vomited. Diseases belonging to this category are chronic gastric ulcer, in which the vomiting commonly occurs at the height of the pain, gastritis, and cirrhosis of the liver, in which morning vomiting may occur, cancer, in which vomiting is not so prominent a symptom as in ulcer unless a pyloric tumour is present.

(*c*) It has been mentioned that very rarely gastric hæmorrhage may result from prolonged vomiting.

(*d*) Gastric hæmorrhage, although profuse and sufficiently severe to kill the patient, may not give rise to vomiting at all. The blood is all passed per rectum as melæna. Such cases are extremely difficult to diagnose.

(5) *The presence of pain and tenderness* usually indicates that the bleeding is due to stomach disease and not to toxæmia. I may here mention that in my experience the presence of multiple points of hyperæsthesia in the skin more often indicates a purely nervous disturbance than actual organic disease of the stomach.

The pain in a typical case of chronic gastric ulcer comes on after food at varying times up to half or three quarters of an hour; vomiting is set up, which puts an end to the pain, which otherwise stops when the stomach is emptied *viâ* the pylorus. It may, however, occur on an empty stomach, in attacks of gastralgia, and at night. It is more severe after solid food than liquids and after very cold or very hot food. The tenderness occurs in the mid-line near the xiphoid process, but lower down if gastroptosis is present; behind it is felt on the left side of the spine on a level with the tenth or twelfth vertebra. Probably this state of affairs is only seen in about half the cases of ulcer or less, although, as I have said before, I think chronic gastric ulcer is diagnosed more often than it exists.

The pain in duodenal ulcer is said to occur from one and a half to two hours after the ingestion of food; there are usually no stomach symptoms at all, and melæna is more characteristic than hæmatemesis. Rigidity of the muscles on the right hand side of the abdomen may be present.

In carcinoma of the stomach there is no particular type of pain. It may be quite absent, sometimes it may be dependent on meals, but often is quite independent of them. It may disappear and recur again, it may be constant, and paroxysms of gastralgia may occur. Tenderness is less frequent than in ulcer as a rule.

In gastritis discomfort after food is commoner than pain, which is not so severe as in the two former diseases. Lancinating pains, which are usually independent of food, are said to occur in atrophic gastritis.

(6) *The physical examination of the patient's abdomen* should be directed towards the ascertainment of the presence of pain and tenderness, dilatation of the stomach, or the presence of a tumour.

It is very important to recognise the fact that perigastritis in cases of ulcer may be so marked as to give rise to a tumour simulating carcinoma. Enlargement of the liver and spleen and the signs of portal obstruction must then be looked for.

The presence of wasting, jaundice, and signs of the various general diseases mentioned above as causing gastric hæmorrhage are to be noted.

(7) About the *general history* of the case nothing need be said, as all the points to be observed may be gathered from what has been previously stated.

It should never be neglected, as it is often of the greatest help in diagnosis.

March 29th, 1909.

Death from the Electric Current from Turning on an Electric Light.—F. E. Jones relates the case of a healthy young man, æt. 21 years, who worked in a machine shop tending a small forge. He stood on a gravel floor mixed with considerable iron chips. Over the forge hung a 16-candle-power, 110-volt incandescent lamp. He reached to turn the light on, uttered a sudden cry, was found stiff and rigid, with one hand clutching the lamp, and leaning against certain iron bars. The contact being broken, he fell to the floor. It was supposed that death was instantaneous. An examination of the wiring system supplying the light showed that the wire to the lamp had come in contact with a wire carrying a voltage of 2300 in connection with a street car system near by. The fuse of the lamp was in good order. These fuses are made of soft metal which breaks when a sufficient amperage goes through them. It is said that the soft metal ages and becomes less resistant, allowing electricity to pass through without burning it out. It only takes from four to five amperes to kill a man.—*Medical Record*, vol. lxxv, No. 11.

TWO CASES OF HÆMORRHAGE INTO THE TESTICLE.*

By V. WARREN LOW, F.R.C.S.

A BOY, æt. 19 years, came to the Out-patient Department of St. Mary's Hospital on April 19th, 1907. He complained that two days previously his left testicle had become swollen and painful. He attributed this, at the time, to the fact that his trousers were too tightly braced up. When this was remedied the pain ceased. There was no evidence of any gonorrhœal infection, but a provisional diagnosis was made of gonorrhœal epididymo-orchitis. A week later he was seen again. The pain, tenderness, and swelling of the testicle still persisted. A most careful examination failed to discover any urethral infection, and he was sent into hospital.

The scrotum on the left side was red and œdematous, the testicle and epididymis were enlarged, and it was difficult through the œdematous scrotum to distinguish one from the other. The vas was normal, and nothing abnormal could be felt in the prostate and vesiculæ seminales. There had been no history of mumps, and he presented no other evidences of tubercle, nor was there any history of tubercle in his family. The case was considered to be one of tuberculous disease of the testicle and epididymis, and the removal of the organ was advised. This was carried out on May 7th, and the patient made an uneventful recovery.

On examining the testicle, which was of a grey-black colour, it was evident that there had been considerable hæmorrhage into the substance of the organ. The attachments of the testicle were normal, and no evidences of torsion could be found. No traces of tubercle were present.

Dr. Spilsbury kindly examined the specimen for me, and I append his report.

The testis and epididymis were enlarged. The digital fossa was well marked; there was no displacement of the epididymis on the body of the testis.

On section the body of the testis was firm and dry, and had a uniform grey colour. In the region of the rete testis were several small ill-defined hæmorrhages. In the epididymis and cord were dilated blood-vessels, but no hæmorrhages were here present, and no indication of torsion of the cord.

Microscopically examined, the cells of the tubules in the body of the testis were completely necrotic, and there was only fragmentary nuclear staining. The same change had occurred in the interstitial tissue, which was everywhere rather wider than normal. The blood-vessels were dilated, and many of the veins were occupied by thrombi.

In the rete testis necrosis was less advanced, and in places nuclear staining was retained; the interstitial tissue was here infiltrated with red blood-corpuscles, and in places there was infiltration also with polynuclear leucocytes.

The arteries of the testes were everywhere patent, and there was no disease of their walls. At the margin of the testis was a hæmorrhagic zone, outside which was a wide zone of granulation tissue, showing all stages in organisation. In places the granulation tissue extended for a short distance into the interstitial tissue of the body of the testis.

The epididymis showed no alteration in structure.

The cord showed dilation and thrombosis of some of the veins.

N.B.—The degree of necrosis varied in different parts of the testis, and appeared to have occurred at different times.

The second case was that of a young man, æt. 21 years, who was sent to me in May, 1907, under the following circumstances: About four or five weeks previously, in the early part of April, he had occasion to get out of bed in order to unlock the door of the room for his brother. While moving, he was seized in his left testicle with a sudden violent pain, of such severity as to cause him to lie on the floor. The pain partially subsided, but all night his testicle was tender, swollen, and aching. On the next morning he was seen by his doctor, who found the organ swollen and tender, and the scrotum red and œdematous. He was kept in bed for a week, and anodyne lotions were applied. The pain and tenderness subsided, and he was much better, but the testicle still remained swollen.

I saw him on May 11th, about four weeks after the attack. His left testicle was then enlarged, and slightly, but not excessively, tender. There

* Delivered before the Medical Society of London.

was no redness nor œdema of the scrotum. Both the epididymis and testicle appeared to be enlarged, and, at the time, I thought the enlargement was more marked in the epididymis, which was hard and seemed to be nodular. There was a general thickening of the cord, but no definite nodule could be felt on the vas. There was no enlargement of the vesiculæ seminales, nor of the prostate, nor was there present any vesical pain or irritability. There was not the slightest suggestion or suspicion of gonorrhœa.

The patient himself appeared otherwise perfectly healthy. There was, however, a bad family history, and the patient was one of the five survivors of a family of eleven, of whom two had died of tubercle.

Under these circumstances, I came to the conclusion that the case was one of tuberculous epididymitis, and advised that the organ should be removed.

This was accordingly done on May 28th. The operation presented no feature of interest, and his convalescence was uneventful.

On examining the testicle, it at once became very dubious whether the case was one of tubercle. There were no deposits in either the testicle or the epididymis that appeared to me to correspond with the effects of a tuberculous invasion, and I therefore submitted the organ to Dr. Spilsbury for examination. The anatomical relations of the cord, epididymis, and testicle were normal, and there was no evidence of any recent torsion of the former.

Dr. Spilsbury reported as follows:

Naked-eye appearances.—The body of the testicle was not enlarged, the epididymis was slightly enlarged, and the digital fossa was deep; there was no displacement of the epididymis upon the body of the testis.

On section, the body of the testis was firm, and had a caseous appearance throughout; there was slight brownish discoloration in the region of the rete testis. The epididymis and cord contained dilated blood-vessels.

Microscopically.—In the body of the testis all the tissues were completely necrotic, and both cell-outlines and nuclei had gone everywhere. The interstitial tissue was somewhat widened. At the margin of the necrotic area was a wide zone of granulation tissue, with newly-formed fibrous tissue. The granulation tissue in places was invading the interstitial tissue of the body of the testis. Some of the granulation tissue-cells were crowded with pigment granules.

In the rete testis were thrombosed veins, and blood-clot almost completely discoloured.

The arteries of the testis were contracted and empty, but were patent, and showed no disease of their walls. There was a little diffuse hæmorrhage into the tissue of the cord, and some of the veins were dilated, and occupied by thrombi.

I have ventured to record these two cases, as they appear to me to be almost identical in nature. In each case there had been a sudden hæmorrhage into the substance of the testicle, with consequent destruction of its glandular elements, and in neither case was the cause of this hæmorrhage at all obvious. Sudden hæmorrhage into the testicle, when it is not an accompaniment of a new growth, is almost invariably the result of torsion, either of the cord or of the "mesentery" of the testicle. Surgical literature of late years is rich in instances of this accident, and I need only refer to recent papers by Edington, Keith and Goring Dowden, Corner, and by Rigby and Howard. But in the cases I have brought forward to-night no evidence of such a twist could be discovered, and, what is, perhaps, of more importance, the testicle and epididymis were normally attached to the back of the tunica vaginalis. In the normal organ the whole length of the posterior border of the testicle and epididymis is bound firmly to the posterior aspect of the tunica vaginalis by a mesentery formed by the reflection of the serous membrane from the viscus to the parietes. The two layers of this mesentery are widely separated from each other by a quantity of fibro-muscular tissue, which is continued down to the lowest part of the sac, and represents the remains of the gubernaculum. It is difficult to conceive how, with the normal attachment, there could be torsion of either cord or testicle. In all the recorded cases of this condition, whenever this point has been investigated, some abnormality of attachment of the testis to the cord has been found. I am therefore at a loss to account for the cause of the hæmorrhage in these two cases, and, as far as I have been able to search the literature of the subject, I have not been able to find any cases which would throw any light on this matter.

Scudder, in a very exhaustive paper in the 'Annals of Surgery' for 1901 (vol. xxxiv, p. 234), quotes a case published by Volkmann in 1877, in which there was spontaneous gangrene of the testicle with acute hæmorrhagic infarction, and in which no twist of the cord was evident. This occurred in a boy, æt. 15 years, who, without any apparent cause, was suddenly seized with severe abdominal pain, diarrhœa and vomiting. On the next day the left side of the scrotum was found to be swollen, and the pain was limited to the left testicle. Three days after the onset the scrotum was swollen with hard inflammatory œdema to twice the size of a man's fist. The left side was of a deep red colour, and was tender and hot.

An incision was made into the tunica vaginalis, and the testicle was found to be swollen to four or five times its normal size, and to be blue-black in colour. The wound was left open, and the testicle and epididymis became necrotic, gradually dried up and dropped off.

Although no mention is made of any torsion, it is stated that there was a long mesorchium, by which I presume is meant the common mesentery of the epididymis and testicle.

Scudder also quotes two cases, published by Englisch in 1893, of hæmorrhagic infarction of the testicle and epididymis.

The first case was that of a boy, æt. 16 years, who in the night had a sudden swelling of the left side of the scrotum without any known cause. The testicle and epididymis became enlarged, the infiltration extending in the cord up to the inguinal canal. The parts were tender and the skin was reddened. The symptoms gradually disappeared and no operation was undertaken.

In the second case, a boy, æt. 17 years, felt a sensation of pressure in the left testicle, and some hours afterwards experienced a severe pain. The parts were swollen, œdematous, reddened and tender, the tenderness extending up to the iliac fossa. At the operation it is stated that the testicle was found to be attached to the posterior wall of the sac by a broad mesorchium. The testicle and epididymis were bluish-black in colour. The tunica vaginalis was drained, and the testicle and part of the epididymis ultimately sloughed. Nothing is said in the account of the case concerning any torsion, and it is definitely stated that the cause of the hæmorrhage was unknown.

These cases appear to resemble the two I have described to-night, but it must be borne in mind that they were published at a time when torsion of the cord or of the testicle was not such a familiar condition as it is now, and so it is possible that these were cases of torsion.

With these exceptions I have not come across any records of similar cases, but I cannot pretend that my search has been an exhaustive one.

I have seen other cases which have closely resembled these I have brought forward to-night, but, in the absence of any definite proof of their nature in the shape of an operation, I did not think it would serve any useful purpose to quote them here. Possibly these cases are not very uncommon, and some of the Fellows of the Society may be able to supply the real cause of their occurrence.

Mr. CORNER said Mr. Warren Low's case might be called a medley of diseases of the testicle. Somewhat similar cases had been recorded under different names, such as gangrene of the testicle, necrosis of the testicle, spontaneous necrosis, thrombosis of the spermatic cord, embolism of the spermatic artery. The real origination of those cases was still a matter of doubt. The modern trend of ideas had been to regard them as due to torsion of the cord. In one case which he had no torsion of the cord was found, but microscopically there was seen to be œdema, and that œdema was sharply delineated by a line differing in colour which could be seen with the naked eye, and the cause of that was probably torsion. But he agreed that a torsion of the cord which was suddenly undone again did not account for all such cases. He did not gather from Mr. Low's paper the character of the naked-eye appearances. He had seen cases in which there was definite evidence to the naked eye of extravasations of blood, and he asked whether that was so in Mr. Low's case. If there were not such visible to the naked eye he would tentatively suggest another explanation, namely, one along the lines of acute hæmorrhagic pancreatitis. The cases which he had brought forward might be acute infective necrosis of the testicle, the infecting organism of which was at present unknown, accompanied by small extravasations of blood not visible to the naked eye. As a rule those cases were impossible of diagnosis, although

in some cases one could guess at the condition of things. But there was one clinical point which, if it was present, would help, namely, if the case was the result of torsion of the spermatic cord, and there was a definite hæmorrhage obvious to the naked eye, no translucency was found on applying that test. If, however, the case was one of epididymo-orchitis, whether tuberculous or gonorrhœal, often hydrocele was present and transillumination would show it. If it were necrosis of the testicle there was no hydrocele.

Mr. KELLOCK asked whether, when Mr. Low divided the cord, he found any hæmorrhage into the substance of the spermatic cord when he removed the testicle. It was important in the diagnosis of the case how far up the cord the hæmorrhage went. It was of interest, because one saw from time to time acute inflammation of the epididymis where there was no apparent cause. Gonorrhœa was suspected in many of them, but possibly the affection which had been mentioned might exist on a mild scale, and with a patient suspected of epididymo-orchitis there might be a small hæmorrhage into the testicle. He did not think the diagnosis need be very important in the case under consideration, because the treatment must be the same in any case. He would not agree with Mr. Corner that it was common in gonorrhœa or in tuberculous testicle. In both those conditions he thought hydrocele would be rather rare, and that the presence of it would not help one in coming to a conclusion.

Mr. A. BARKER said he could not say cases which he had seen were similar to Mr. Low's, as he had not operated upon them, but he could recall cases of his own which were very like his. He remembered one very well, that of a healthy man who appeared to have no trace of gonorrhœa. He was walking close to the hospital with a plank on his shoulder when he slipped on a step, and in trying to recover himself ricked his external ring. He entered the hospital within an hour of the accident with a testicle as large as a goose egg, having been very well before. He supposed that the man had ruptured a vessel there, owing to the pressure of the weight he was carrying plus the muscular and expiratory effort occurring with the sudden start. The vein had extravasated into the testicle itself. Another case which occurred soon afterwards was on all fours with that. He did not think Mr. Low had sufficiently emphasised the amount of strain that such patients suffered from. The rapidity of the inflammation in his cases was very marked.

Mr. C. B. LOCKWOOD said the question arose whether the hæmorrhage was due to something which began above in the cord, or whether it was due to something occurring in the testicle itself. He understood the author that in one of his cases the hæmorrhage was into the body of the testes, which seemed to negative the possibility of it having occurred from something above in the cord. He did not understand from Mr. Low that the onset in his cases was particularly acute, which did not seem to bear out Mr. Corner's reasonable supposition that the condition might be due to an infective state of the testicle itself, which would be curious if it was confined to the testicle.

Mr. Low, in reply, said that if he had known exactly what he was going to find he would not have operated. In the second case the testicle was practically fibrotic. There was some granulation tissue which in time would have formed a fibrotic mass. The first case he operated upon earlier, and in that there was definite hæmorrhage. Mr. Corner had kindly referred him to a paper on the subject in the 'Annals of Surgery' for August last, but the cases in that paper did not seem to be parallel to his own. With regard to the naked-eye appearances, the first case was operated upon within a fortnight of the occurrence, and there was definite hæmorrhage, which one could see with the naked eye. The testicle was greyish-black. The hæmorrhage was chiefly at the junction of the testes with the epididymis. In neither of the cases was there hydrocele. There was no visible hæmorrhage into the spermatic cord, but the veins were dilated and probably contained thrombi; at any rate there was blood-clot. Mr. Barker's cases seemed to have closely resembled his own. With regard to the strain exerted, the boy in his case was in the habit of wearing two different pairs of trousers, one of which was long-waisted and the other short-waisted. He forgot to adjust his braces accordingly, and when he felt the pain he loosened his braces, but the pain did not cease and the testicle swelled. In the other case the patient jumped suddenly out of bed to unlock a door to let his brother in, and he felt pain in his testicle from that moment. In both the cases there was rapid swelling, and in a future case he thought he would rely on that point in making his diagnosis, as it would probably differentiate it from an inflammatory swelling.

March 29th, 1909.

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VENOUS THROMBOSIS.*

By F. de HAVILLAND HALL, M.D., F.R.C.P.,
Senior Physician to Westminister Hospital.

THROMBOS is a Greek word signifying a lump or piece with a secondary signification of a clot of blood, so thrombus has been very appropriately introduced as the term to express the formation of a blood coagulum in the heart or vessels during life. The act or process of the formation of a thrombus is called thrombosis.

Thrombosis may take place in the heart, the arteries, the capillaries, the veins, and the lymphatics, but on the present occasion I intend to limit my remarks to thrombi as they occur in veins.

A thrombus is composed of all the elements of blood, *i. e.* it consists of fibrin and red and white blood-corpuscles. In thrombi which are formed rapidly there is a large proportion of red blood-corpuscles, hence the thrombus is of a dark red colour. In the more slowly forming thrombi the red blood-corpuscles may be more or less absent, and the thrombi are consequently mixed red and white, or quite white.

Thrombi have been divided into the laminated and the non-laminated or uniform—the name expresses the difference between the two. After a thrombus has been formed various changes may occur in it. As already mentioned the quickly formed thrombi are of a red colour, but with a process of shrinking the clot loses its colour and may become yellowish-white and tougher in consistence, or may even be friable. By a further process of shrinking the thrombus may become leathery, and as a final stage calcification may occur, producing a calcareous body known as a

* Delivered before the Ealing Division of the Metropolitan Counties Branch of the British Medical Association.

phlebolith. Or instead of becoming harder and firmer the thrombus may soften and break down, and may even suppurate. Suppuration occurs at times in thrombi which are surrounded by an inflamed area.

In a certain proportion of the cases the clot becomes organised.

In none of the cases that I have seen has suppuration occurred.

As regards the immediate cause of the clotting of blood in the veins, several factors have to be taken into account. The old view was that phlebitis, *i. e.* an inflammation of the vein, was the primary condition, and that the clotting was secondary to it. Virchow was thought to have upset this view, and he attributed the presence of thrombi in vessels to four causes: (1) Compression of vessels; (2) dilatation of vessels; (3) marasmus; (4) trauma. All these conditions are to be accepted as contributory factors, but we now recognise that the composition of the blood and the condition of the vein have also to be taken into account. For instance, leucocytosis appears to favour thrombosis. This is probably the explanation of the blocking of veins occurring after hæmorrhage and in states of anæmia. Then, as Sir Almroth Wright* has shown, an excess of lime salts in the blood leads to clotting. What changes the various specific diseases bring about in the blood, and how these cause clotting, is at present unknown. Micro-organisms have been found in thrombi in typhoid cases and also in phthisis, but it is a question whether they appeared before or after the thrombus had formed.

Next we have to consider the vessel wall, and there is much evidence in favour of the view that thrombosis starts with a primary inflammation of the wall of the vein. This seems especially true in respect to varicose veins and in gouty cases. In typhoid fever, too, the vein wall has been found to be infected with typhoid bacilli. Lastly, we have to consider the state of the circulation. That thrombosis is so much more frequently met with in the lower extremities than in the upper seems to suggest that stasis has something to do with the thrombosis. Feeble circulation will also be a contributory factor in cases of anæmia, both idiopathic and that due to loss of blood. One of Dr. Gee's aphorisms* sums up the question in an admirable manner: "Phlebitis is common in phthisis, as indeed it is in many infective diseases. This would seem to favour the view that the condition is a primary one in the veins, and not the result of thrombosis, though this is doubtless also true in some cases. In this connection it should be remembered that suppurative organisms form an important factor in most cases of phthisis."

Thrombosis in the veins of the lower extremity may occur in influenza, typhoid fever, the puerperal state, pneumonia, pleurisy, anæmia and chlorosis; in septic conditions and especially appendicitis, gout and diabetes. It may occur as a result of the varicose condition of the veins, especially if traumatism be superadded. It has happened in a varicose saphena from subcutaneous rupture during violent coughing. Thrombosis is also met with in cases of exhaustion from acute or chronic diseases, as in the last stages of cancer and pulmonary tuberculosis. And, finally, it occurs by extension from inflammation in connection with new growths, ulcers and gangrenous or acutely inflamed parts, or from the pressure of an abscess or tumour.

In all forms of thrombosis the left femoral vein is much more frequently affected than the right; according to Leibermeister this is due to the fact that the right iliac artery crosses over the left common iliac vein, and consequently the flow of blood is not so free as in the right vein. I am disposed to regard the interference to the circulation through the vein from over-distension of the sigmoid flexure as a contributory cause of the more frequent occurrence of thrombosis in the left femoral.

In an interesting analysis of forty-two cases of venous thrombosis occurring in the course of typhoid fever, Dr. Thayer† shows that in forty cases the lower extremities were affected, in one the upper extremity and in one the pulmonary artery. As regards the side of the body: the left was affected twenty-six times, the right five times, both sides nine times, and in two cases the records are imperfect. As regards the vessel affected, the

* 'Med.-Chir. Trans.,' vol. lxxxvi, p. 18.

* 'St. Bartholomew's Hospital Reports,' vol. xxxii, p. 40.

† 'Transactions of Association of American Physicians,' 1904, vol. xix, p. 164.

femoral vein was attacked twenty-one times, the popliteal, iliac and veins of calf each five times, the internal saphenous three times, the pulmonary artery, the pulmonary artery and common iliac, and the axillary vein each once. These figures bear out what I would have said *à priori*, viz. that the left femoral vein is the most common seat of thrombosis. This is certainly the case so far as thrombosis resulting from typhoid fever is concerned, but in the thrombosis or phlebitis—call it what you may—of gouty origin there is no such predilection for the femoral, on the contrary, the veins of the calf are more commonly affected, and I am sure that it is a more generalised affection, more veins are involved at the same time, and as some veins clear up others are attacked.

In my experience influenza is one of the most potent causes of thrombosis, and the cases have been of a grave character. Three are particularly impressed upon my memory, as in all three death occurred with startling suddenness.

The first case was that of a young medical man who had had a sharp attack of influenza. While convalescing he was seized with acute pain in the calf of the leg, and the leg soon became much swollen and very painful, in fact, a typical example of femoral thrombosis. Absolute quiet was enjoined on the patient, and the treatment to which I shall direct your attention later on was carried out. Nothing occurred to give rise to any extra anxiety, but one night, about a week from the commencement of the thrombosis, I was hurriedly summoned to the patient's bedside only to find him dead. I was told that he was apparently in his usual condition when he rather suddenly raised himself in bed and fell back unconscious and died almost at once.

The second case was that of a man, æt. 47 years, to whom I was called by his medical attendant on account of thrombosis of the right popliteal vein following influenza. The patient was cautioned as to the extreme importance of keeping absolutely in the recumbent position, but disregarding our advice he got out of bed some days later, and died quite suddenly.

The third case was quite dramatic in its suddenness. I was asked by a general practitioner to see one of his patients, a man, æt. 45 years, with him. He said that there really was no anxiety about the case as the patient had had a mild attack of influenza, and while apparently convalescing he had got pleurisy with a moderate amount of effusion. As the patient was a very nervous man and very much alarmed about his condition I was asked to do my best to reassure him. I made a careful examination of the patient, and in order to get at his back I sat him up in bed. While examining the patient I somehow or other touched his leg, when he cried out and said that I hurt him. This directed my attention to the leg, and on examining it I found a typical example of femoral thrombosis. Having already had two fatal cases of post-influenzal thrombosis I pointed out to the doctor the grave condition the patient was in, but in view of his extreme nervousness we agreed that it would be best not to alarm him. I therefore passed lightly over the state of the leg, but told him that as he had pleurisy he must keep quite quiet in bed in the recumbent position. I, of course, informed the relations of the patient about the anxious state of affairs. I then left the house, and while talking to the doctor outside we were hurriedly summoned back to the house only to find the patient dying, and death took place within ten minutes from the time we re-entered the house. I have always thought that my sitting the patient up and making him exert himself loosened the clot and brought about the fatal termination.

One post-influenzal case occurred in a lady, æt. 78 years. While convalescing from influenza she had a fall and struck the right buttock. This caused a good deal of pain. Four days later an eruption of herpes came out all down the leg, and a week later the leg was swollen, tense, and shiny, clearly due to femoral thrombosis. This is the only case in which I have seen herpes accompanying thrombosis. The patient made a good recovery.

An exceedingly troublesome and protracted case was that of a lady, æt. 39 years. I saw her originally in 1899 after a very severe attack of influenza followed by desquamation of the cuticle. Subsequently she had another attack of influenza, also followed by desquamation, and for two years after this her temperature went up every day to over 100° F. In 1904 she was operated on for appendicitis, the appendix being removed. The operation was followed by thrombosis in the left femoral vein. In the summer of 1906 she seems to have been quite well, but between September of

that year and May, 1907, she had four attacks of influenza followed by persistent elevation of temperature. In February there was a return of thrombosis in the left leg; this was followed by a block in the right femoral, and when I last heard of her in September, 1907, the legs were still swollen and she was unable to get about. This patient seems peculiarly susceptible to the poisons of influenza, and to get the infection in a very virulent form.

In none of the cases of influenzal thrombosis which I have seen have there been so many relapses as in this case.

Next to influenza as a cause of thrombosis must be placed typhoid fever, but unlike the influenzal cases all the typhoid cases which I have seen have recovered with life, though in most cases with impaired power in the leg.

The attack may come on after the temperature has fallen to normal, so that a rise of temperature during the period of convalescence should awaken the suspicion that thrombosis has occurred. In Dr. Thayer's thirty-nine cases the onset was almost invariably in the third week or later. The rise in temperature is usually only slight, but occasionally the temperature may rise to 104° or 105° F. Fever is a very constant symptom, being found in thirty-six of the thirty-nine cases. In twenty-six cases the fever was intercurrent, the thrombosis coming on before complete defervescence. In thirteen the temperature was normal at time of onset.

The patient invariably complains of pain, which is frequently severe. It is sometimes first noticed in the calf. The pain may persist for days. In the five cases of iliac thrombosis recorded by Dr. Thayer the onset was associated with sudden sharp abdominal pain in four instances. In one of these cases the pain was very severe, and as there was also leucocytosis, perforation was suspected and an exploratory laparotomy was performed. Chills are present in rather more than a quarter of the cases, so that a rigor during convalescence from typhoid should certainly suggest the possibility of thrombosis.

Swelling of the limb is present in every case, and is sometimes so great as to cause the affected limb to be several inches greater in circumference than its fellow. In about half the cases, in addition to the swelling, there is œdema. On examining the leg it may be possible to detect the thrombosed vein as a hard cord, but usually it cannot be distinguished.

Dr. Sydney Phillips* has recorded a case of thrombosis of the inferior vena cava and of the iliac and femoral veins, of acute onset and fatal termination, with symptoms resembling those of perforated gastric ulcer.

Most valuable and suggestive information as to "the causation and treatment of thrombosis occurring in connection with typhoid fever" is contained in a paper with the above heading communicated to the Royal Medical and Chirurgical Society by Dr. (now Sir) Almroth Wright and Dr. Knapp.† From their researches it is shown that during the acute stage of the disease there is a general diminution of blood coagulability. This diminished coagulability furnishes an explanation of the serious hæmorrhages which may occur from comparatively small lesions in typhoid fever. Dr. Wright and Dr. Knapp also show that the quantum of lime salts in the blood of the typhoid convalescents examined was greatly in excess of that in normal blood. From this fact they argued that the increased coagulability during the convalescent stage may be dependent upon an excess of lime salts, and they point out that this excess of lime salts must be derived from the milk which usually constitutes the exclusive dietary of typhoid patients. From this they deduce the indications for the prophylaxis and after-treatment of thrombosis, both when it occurs in connection with typhoid fever and when it occurs in connection with other diseases. This indication is met by the administration of citric acid, which should be given as soon as the danger of intestinal hæmorrhage has passed away. The best plan is to add twenty to forty grains of citrate of sodium to every pint of milk. This addition has the advantage of rendering the milk more easily digestible, and of preventing constipation.

Both in hospital and private practice I have seen numerous cases of femoral thrombosis following typhoid fever, but only one of the cases presented any unusual features. This was a man, æt. 34 years, who was sent to me in June, 1903, on account of persistent swelling of the leg. The history I received of the case was that he had had a very long but not very severe attack of typhoid fever

* 'Clin. Soc. Trans.,' vol. xxxiv, p. 24.

† 'Med.-Chir. Trans.,' vol. lxxxvi, p. 1.

starting on January 27th, and that he kept his bed until the middle of April. During convalescence he developed thrombosis of the left femoral vein. This nearly cleared up, though there was still some swelling of the ankle when he went away for change. Six weeks later, on his return home, both legs were found to be œdematous, and the veins at the sides of the abdomen were enlarged. On examining the patient I agreed with his medical adviser that there was evidence of the thrombosis having extended up to the veins in the abdomen. The last report I received of the patient was in January, 1904, when I was informed that the legs still swell and the superficial abdominal veins remain large.

That anæmia pure and simple is capable of producing thrombosis has long been known, but I do not remember myself to have seen a case until a patient was admitted, on December 20th, 1907, into Queen Adelaide Ward, Westminister Hospital. The patient was a woman, æt. 23 years, by occupation a housemaid. Her family history is unimportant. The patient states that she has had no previous serious illnesses, but has always been pale. She first noticed pain in her right thigh on December 12th; this gradually spread to the knee, where it has remained. On the 16th she noticed swelling on the inner side of the thigh, accompanied by great tenderness.

On admission the patient was very anæmic. She complains of pain along the inner aspect of the right thigh, here there is a great tenderness, and a cord-like structure is palpable in the course of the femoral vessels; there is some œdema about the ankle.

Pulse 76. Systolic murmur heard all over cardiac area, but loudest in the pulmonary area. Bruit *de diable* is present.

On December 27th patient complained of severe cramp in right leg and pain in left axillary region, but on examination no evidence of pulmonary mischief was found.

On January 3rd it was noted that the right thigh and leg were very much swollen, but there was now no tenderness or pain.

By January 10th the patient was very much better. The thigh and leg are much reduced in size. A patchy rash of a pale red colour, with ill-defined margin, not raised, has appeared over the lower fourth of the leg and ankle. The skin is uniformly glistening. On January 17th the patient had still further improved, the thigh and leg had returned nearly to the normal condition, and there was no pain or tenderness. Pulse 74, of fair volume. The cardiac murmurs are much less distinct.

It has only to be added that two blood counts were made. The first, the day after admission, showed—hæmoglobin 38 per cent., red blood-corpuscles 4,060,000, white blood-corpuscles 8000; and the second on January 17th showed—hæmoglobin 53 per cent., red blood-corpuscles 4,710,000, white blood-corpuscles 9400, the examination of the blood thus confirming the diagnosis of chlorosis which had been arrived at, but there was not the leucocytosis one would have expected.

The late Dr. Tuckwell, in an able article in vol. x, 1874, p. 33, of the 'St. Bartholomew's Hospital Reports' "On Clotting of Blood in Gout and Chlorosis," describes a case of thrombosis as it occurs in the femoral vein of a chlorotic girl. He raises the question whether some of the cases of dropsical feet and legs in chlorosis, stated in books to be caused by a too watery state of the blood, are not really due to true venous obstruction.

He also describes a case of thrombosis of the cerebral sinuses and veins occurring in a chlorotic girl, æt. 16 years, verified by a post-mortem examination. I have not myself seen a similar case. It is important to remember the possibility of the occurrence of cerebral thrombosis in chlorosis, as it is a cause of death in a disease which usually tends to recovery. The anæmia produced by profuse hæmorrhage is one of the causes of femoral thrombosis. I had an interesting example of this in a young woman who was under my care in Queen Adelaide Ward, Westminster Hospital, at the end of 1903. She was admitted quite blanched from loss of blood, consequent upon a gastric ulcer, and while convalescing she developed femoral thrombosis. She made a good recovery, both from the original malady and also from the complication. While she was still under treatment a man, æt. 25 years, was sent to me with a clear history of intestinal hæmorrhage due to duodenal ulceration. As he was convalescing from this he got some deep thrombosis in his left leg. He also made a satisfactory recovery.

One of the most severe cases I have seen of

femoral thrombosis occurred in a young lady, æt. 17 years, who was under my care in a nursing home. She was admitted for operation on account of appendicitis, but when she was on the operating table under an anæsthetic the tumour, which had been distinctly palpable the day before, had disappeared, so the surgeon decided not to operate. The abscess probably discharged through the bowel, as puriform matter was seen in the stools. The patient was hardly out of danger from the appendicitis when she developed a very acute attack of thrombosis in the left femoral vein. The leg became very painful and enormously swollen, and nearly two years elapsed before the leg could be pronounced fairly well.

Pneumonia is occasionally followed by thrombosis of the femoral vein. I saw a case of the kind in consultation with Dr. Godson in March, 1903. The patient was a lady, æt. 22 years, who had a perfectly normal first confinement, and two days later was attacked with lobar pneumonia. This lasted a week, and just as she was convalescent she complained of pain in the left groin; the leg became rapidly swollen and showed all the signs of typical femoral thrombosis. It may be argued that this was a case of phlegmasia dolens of septic origin due to the previous delivery, but the leg presented the appearances met with in ordinary thrombosis and did not look like the leg of phlegmasia dolens. The patient made a good recovery, though the leg still remained a little swollen when I saw her in the end of July of the same year.

I have notes of three cases of thrombosis following pleurisy, and if I had thoroughly searched my case books I think that I should have found more.

The first case I saw in a private asylum in December, 1897. The patient was a lady, æt. 33 years, suffering from dementia. When I examined her I found a pleural effusion on the right side with signs of blocking in the left femoral vein, and I ascertained that three weeks previously the right leg was swollen. Both legs gradually cleared up, but in the middle of January, 1898, the swelling of the legs came on for a few days, but by the end of the month she was physically well. The other two cases occurred in connection with an attack of pleurisy following parturition, so that, as in the case of pneumonia I have just mentioned, the thrombosis may have been due to puerperal mischief and not be the result of pleurisy. The first was the case of a lady, æt. 32 years, who was confined on January 24th, 1901. On February 21st she went downstairs and got a chill, and was seized with acute pain radiating all over the right chest. When I examined the patient on February 25th I found dulness up to the angle of the scapula and defective entry of air. While convalescing from the pleural effusion she developed thrombosis, but eventually made a good recovery. In the remaining case a lady, æt. 38 years, had had trouble with varicose veins three or four weeks previous to her confinement. Four weeks after her confinement she had pleurisy on the right side, and a week later pain, tenderness and swelling in the right leg. This patient also did well. I have narrated the histories of one case of pneumonia and two cases of pleurisy following parturition which were subsequently accompanied by thrombosis of the femoral vein; this would seem to suggest that the occurrence of pneumonia or pleurisy in a puerperal patient should awaken the suspicion of the possibility of thrombosis coming on.

That gout is one of the causes giving rise to thrombosis I have no doubt, but whether the thrombosis is the primary condition is more difficult to decide. In fact I do not think that we are in a much better position to do so than when Sir James Paget in his classical 'Clinical Lectures and Essays,' wrote as follows: "Of many of them, indeed, I cannot tell, any more than of certain other forms of phlebitis, whether the inflammation or the clotting were the first event, nor, therefore, whether they are to be referred more properly to phlebitis or to thrombosis. But I give the former name to them because it is in most common use amongst us and is probably correct for at least one part or stage of every case."

He goes on to say of gouty phlebitis: "The use of this name is, I believe, justified by the frequent occurrence of cases in which phlebitis either is associated with ordinary gouty inflammation in the foot or joints, or occurs with little or no evident provocation in persons of marked gouty constitution or with a gouty inheritance."

At the present time, as I have already mentioned, there appears to be a tendency to revert to the old view of a primary inflammation of the walls of the vein, which was supposed to have been overthrown by Virchow. The gouty cases seem to belong to a

different category to the typhoid and influenzal cases. The superficial veins are first affected and the mischief occurs in patches. Most commonly the veins of the lower extremity are attacked, though occasionally the veins of the trunk and arms are similarly affected. From the superficial the clotting may extend into the deeper vessels. The thrombosis may occur in healthy veins or in those which are varicose, and it is just in the latter case that the thrombosis is particularly liable to occur after an injury or strain. Sir James Paget* held the view that gouty phlebitis was often hereditary.

I saw in November, 1901, in consultation with Dr. Coates, of South Kensington, a typical example of what Sir James Paget would have called gouty phlebitis. The patient was a vigorous man, æt. 80 years, still leading an active, busy life, but he had been subject to gouty attacks. He had been confined to the house for two or three weeks with bronchitis and some congestion of the base of the right lung. Ten days before I saw him he had slightly injured his left leg. There had been pain and tenderness for a week, and when I saw the patient the leg was much swollen, but not very painful. The leg gradually improved under treatment, but when the patient was allowed to get out of bed the right leg started off and he had all the old trouble over again. The tendency to relapse is one of the most characteristic features of gouty thrombosis; just as the swelling appears to be clearing up another vein gets blocked, or the opposite leg becomes affected as in the case I have just mentioned.

A case I saw with Mr. Jago on November 26th, 1906, well illustrates this tendency to relapse and also the risk of detachment of emboli. The patient was a man, æt. 53 years, who on June 20th struck his right shin and had pain in the calf of the leg. This went on for five or six weeks. On October 20th he had another attack, the right leg became an inch larger in circumference than the left, and there was some redness over the inner aspect of the thigh. The temperature was but little raised, going up to between 99° F. and 100° F.

I saw the patient again on December 17th. I found him looking very ill, slightly cyanosed, with embarrassed breathing and a pulse of 120 per minute. I heard that four days previously he had experienced sudden acute pain in the right side of the chest followed by sanguineous expectoration and signs of fluid in the chest. His temperature was not much elevated. I visited the patient again on December 29th and heard that he had had an attack that morning similar to the former one, except that this time the pain was in the left chest. On examining the patient as well as I could without disturbing him I heard a pleural creak in the left infra-axillary region, and there was dulness at the right base. The pulse was 120 per minute, and the patient's condition gave us great anxiety. In spite, however, of these two alarming attacks of pulmonary infarction, the patient eventually made a good recovery. The persistence of the trouble and the number of relapses which occur is well exemplified by a case under my care in the Hostel of St. Luke last year, to which I shall refer later on.

As in the other forms of thrombosis, so in the gouty cases pain and swelling are the two most prominent symptoms. In some instances the pain is of an agonising character, and the swelling only moderate in amount, and in others the swelling is very pronounced and the pain only slight. In one attack a patient may experience much pain and little swelling, and in the next much swelling and little pain; what determines the difference I cannot say.

Among the cases of thrombosis which I cannot assign to any very definite cause was that of a lady, æt. 22 years, whom I saw in consultation with her medical adviser. I was given a history of gastric ulcer, *i.e.* pain after food and vomiting. Eleven days before I saw her while she was out in a bath-chair she was suddenly seized with intense pain in the left lumbar region and diarrhœa. The temperature was from 103° to 104° F. For two or three days previous to my visit the temperature was about 100° F. On examination I found a tender swelling in the left lumbar region, which, I have no doubt, was an abscess resulting from perforation of the stomach. The left leg was œdematous, the swelling having started the day before my visit. Operative interference was declined, and the case terminated—as almost all unoperated cases of rupture of the stomach do—fatally. This may possibly be regarded as thrombosis of septic origin.

* 'Clinical Lectures,' p. 304.

A condition which requires to be differentiated from simple thrombosis is phlegmasia dolens. This is a swelling of one or both legs usually coming on a short time after delivery, but rarely before the second week. Of this disease Dr. Tyler Smith said: "I look on a woman attacked with phlegmasia dolens as having made a fortunate escape from the greater dangers of diffuse phlebitis or puerperal fever." The disease is essentially a parametritis, and the inflammatory mischief extends from the womb along the planes of cellular tissue, causing phlebitis and lymphangitis with secondary thrombosis. The appearance of the leg in phlegmasia dolens differs from that seen in simple thrombosis inasmuch that the leg is whiter—hence its old name of "white leg"—harder and more brawny, not pitting on pressure. Besides being pale it is also glossy or shiny, and the inflamed lymphatics can be seen as faint red lines.

Prognosis.—The prognosis in femoral thrombosis is always an anxious one. Curiously enough the only fatal cases I can remember are the three I have recorded as being due to influenza and one of gouty origin, hence I think that we must assume that there is some extra risk attaching to cases of thrombosis occurring as a result of influenza. The imminent risk of femoral thrombosis is a detachment of a portion of the thrombus with pulmonary embolism as the result. This apparently happened in all the three fatal cases I have recorded as due to influenza, and in all three death occurred in from five to ten minutes from the onset of the fatal symptoms. In thirty-nine cases of venous thrombosis recorded by Dr. Thayer, which occurred while the patient was under observation for typhoid fever, death took place in five, *i. e.* a mortality of 12·3 per cent., but in only two instances was the thrombosis the direct cause of death, once from pulmonary embolism and once from embolism of the inferior cava and right auricle. "Equally with the other forms of phlebitis, but as rarely as in any, that which occurs in gout may be fatal or very dangerous by embolism"; so writes Sir James Paget. *

Should a large piece of thrombus become detached, causing pulmonary embolism, the patient is in a state of urgent dyspnœa, has an anxious expression, is of an ashy-pale colour, extremities cold, and body covered with a clammy sweat. The pulse is very frequent and hardly perceptible. Consciousness is usually maintained until nearly the end. Where only small pieces of the thrombus are detached they enter the lungs, causing infarcts. This occurred in two of the cases I have brought before your notice to-day. Pulmonary infarction is usually signalised by an attack of dyspnœa, acute pain in the chest, followed by cough with bloody expectoration. On examining the chest a patch of dulness with pleural friction may be detected. There is a rise of temperature and increased frequency of the pulse. The danger of the detachment of emboli is greatest during the first week or ten days; after a month from the onset there is very little risk, but it is best to be on the safe side and keep the patient absolutely quiet for six weeks from the commencement of symptoms of thrombosis.

So much for the prognosis as regards life; now we have to consider prognosis in respect to the condition of the leg. In the first place the duration of the acute symptoms is most uncertain. In a case of moderate severity the leg remains painful and swollen for two or three weeks; then the swelling gradually begins to subside, and at the end of another two or three weeks the leg may have returned to its normal size, but when the patient begins to get about again there is usually a certain amount of œdema. Unfortunately this comparatively favourable course has many exceptions. You will have gathered from the histories of the cases I have given you that thrombosis has a great tendency to relapse, one part of the leg will seem to be improving, and then some of the deeper veins get blocked, or possibly the other leg may become affected, so that when a patient is laid up with femoral thrombosis it is unwise to venture upon any prediction as to when he will be able to leave his bed. Even when the patient has recovered sufficiently to enable him to walk about the leg may remain enlarged, and readily swells on slight exertion. Occasionally a leg in which femoral thrombosis has occurred never returns to its normal size, and may seriously interfere with the patient's power of locomotion. In one case to which I have directed your attention there was apparent extension of the thrombosis up to the veins in the abdomen, and a year after the onset of the trouble the legs were still swollen. I

* 'Clinical Lectures,' p. 303.

have recently heard the after-history of two of the cases I have described. The first one is the case of recurrent thrombosis after influenza and appendicitis. The patient wrote to me in September, 1908, fourteen months after my last visit to her, saying that though she was not quite well she was able to walk half an hour or forty minutes at a time. Considering the number of relapses the patient had and the severity of the attacks this is a much better report than I expected, and should warrant one in speaking hopefully to patients. The other case was that of a young lady with appendicitis, in whom the abscess discharged through the bowel. Her mother writes to me: "There is considerable difference between the two legs, the arch of the foot has largely given way and the foot turns out more than the other one; the leg was bandaged continuously for about three years, but for the last year or more she has given up her bandage, and the leg gives practically no trouble, although it makes her walk in rather an ungainly way." Dr. Thayer gives a very unfavourable report; he says that in all instances there has been more or less permanent disability, among the after-results being cramps, pains after standing, œdema and permanent enlargement of leg, varices and a feeling of weakness in leg. Dr. Osler in the discussion following the reading of Dr. Thayer's paper took a more hopeful view; he said: "I have known a number of cases in which the recovery has been perfect, and I think our hospital experience is exceptional." Dr. Thayer's paper was based on cases in Dr. Osler's wards.

A patient who was under my care in the Hostel of St. Luke during last autumn is an excellent illustration of the chronicity and tendency to relapse in gouty cases. The patient, a clergyman, æt. 51 years, was attacked with a sharp pain in the calf of the right leg in December, 1907. Since this time, with the exception of the month of June, first one leg and then the other has been attacked, so that he has been almost constantly confined to bed. In the later attacks the veins of the abdomen and gluteal region have been affected. The veins implicated have been invariably the superficial ones, and their course has been marked by redness, hardness of the vein and tenderness. Later (November 14th) he was just recovering from inflammation affecting the superficial veins over the outer aspect of the right thigh. On the night of November 21st he was seized with acute pain in the abdomen and passed a considerable amount of blood, and died shortly afterwards. From the symptoms I infer that he had thrombosis of the mesenteric veins with hæmorrhage into the bowels. Unfortunately I could not obtain a post-mortem examination.

One of Dr. Tuckwell's* cases shows that recurrence may take place even after years of immunity. The patient, a man, æt. 60 years, had a very severe attack of thrombosis in the left internal saphenous vein attended with pulmonary embolism in September, 1864. In November he was still very ill, and expectorating blood, but after an attack of gouty inflammation in the hand he eventually completely recovered. He lived six years after this, a fine strong man, till November, 1870, when he had another illness just like the former one. About six weeks from the beginning of this illness he died from an attack of pulmonary embolism, after having had a milder attack of embolism This history will lead one to give a guarded prognosis in cases of gouty thrombosis. On the other hand Mr. Prescott Hewett† in speaking of prognosis in cases of gouty thrombosis, wrote as follows: "I have known one gentleman who, after an attack of this kind, which confined him to his couch for months, returned to deer-stalking with as much zest and activity as before."

A case recorded by Dr. Francis Hawkins,‡ of Reading, in which death occurred from cerebral embolism, illustrates the tendency there is in some instances for thrombosis to occur in many parts of the body. In this case, at the necropsy, there was venous thrombosis in all four extremities, and thrombosis on the posterior cusp of the mitral valve, the latter being evidently the origin of the embolus carried to the brain. In the same communication Dr. Hawkins reports a case of thrombosis in the left lower limb, followed by gangrene, amputation, recovery, but the evidence does not convince me that the gangrene was the result of thrombosis, and, unfortunately, the vessels of the amputated limb were not examined.

Treatment.—From the history of the cases and the account of the prognosis I have given, it will be at once seen that absolute rest in bed is of

* 'St. Bartholomew's Hospital Reports,' vol. x, p. 25.

† 'Transactions of Clinical Society,' vol. vi, p. 40.

‡ 'Lancet,' 1904, vol. i, p. 796.

primary importance. The patient should be placed on a spring bed with a firm mattress, and with one pillow for the head. The affected leg may be elevated on pillows and protected by a cradle, but a better way of securing the complete immobilisation of the limb is to employ a Laurence's back splint swung on a cradle. Wooden side splints should be used and the splints well padded. The splints should be kept in position by webbing fixed with a buckle, so that the limb can be conveniently inspected and applications made to it if necessary. The great advantage of having a back splint slung from a cradle is that it facilitates the use of the bed pan without disturbing the patient unnecessarily, and also allows him a certain amount of movement, which is a great consideration when the length of time that the patient will be confined to bed is borne in mind. The history of two of the cases I have recounted will emphasise the necessity for absolute rest, because I believe that in one of these cases sitting the patient up for examination was the probable cause of the fatal termination, and in the other case the patient suddenly sitting up in bed seemed to have caused death. The diet of the patient should be light and easily digested. As long as there is any febrile disturbance it should be liquid, about three pints in the twenty-four hours, one pint of beef-tea to two pints of milk, five ounces being given every two hours. When the temperature comes down lightly boiled eggs, fish, poultry, game, sweet-bread and a little mutton or lamb may be given. Alcohol is best avoided. During the early stage the medicinal indications are confined to quieting the patient's nervous system, and to the relief of pain. If the patient be very restless some bromide may be given. For insomnia sulphonal, trional, or veronal may be ordered. The free application of a pigment composed of equal parts of the green extract of belladonna and glycerine over the affected part will usually give great relief. If the leg be hot and inflamed lead lotion with or without tincture of opium may be employed with advantage. If the pain be very severe it may be necessary to employ a hypodermic injection of morphine and atropine; one sixth of a grain of morphine sulphate with one two-hundredth of a grain of atropine sulphate is usually sufficient, but of course larger doses may be required. If the pulse be very frequent tincture of strophanthus may be ordered. The bowels should be kept free by an occasional dose of calomel or compound senna mixture.

Should any source of infection have been discovered this should be treated, as, for instance, in cases of thrombosis coming on in patients with discharge from the vagina warm antiseptic injections should be used. After the acute stage is over it may be necessary to adopt some general treatment. In cases of thrombosis coming on after loss of blood or in connection with anæmia, iron with or without arsenic is needed. As these patients are often constipated a combination of sulphate of iron and sulphate of magnesium is generally very useful. In gouty cases iodide of potassium, chloride of ammonium and alkalies will be found to answer well. These patients may also drink Vichy water. In view of the tendency of milk to promote clotting of blood I have recently ordered citrate of sodium, one grain to the ounce of milk, in all patients suffering from thrombosis. As a measure of precaution this plan might be tried in all cases where the patient is confined to bed on a milk diet. This method of treatment is especially indicated in cases of typhoid fever after the ulcerative stage is over, owing to the frequency with which thrombosis occurs as a sequel to typhoid fever.

The duration of the immobilisation of the affected limb depends partly upon the severity of the case, but in all instances I insist on a minimum of three weeks after the last elevation of temperature, or about five or six weeks from the commencement of the thrombosis. After taking off the splint the leg should be left alone for four or five days, but about the sixth day passive movement of the foot should be commenced. Any elevation of the temperature should lead at once to cessation of the movements. The next step is to educate the veins by allowing the leg to be placed in a dependent position for a few minutes. After a few days the patient may put his feet to the ground. At this stage of procedure gentle stroking (effleurage, as it is technically termed by professors of the art of massage) of the limb should be commenced, but anything like massage must be avoided on account of the risk of detaching an embolus. Gentle effleurage may be repeated daily for five or ten minutes. By its means the cutaneous circulation is improved, the œdema lessened and duration of convalescence diminished. In practising effleurage

care must be taken to avoid the vein. Before the patient is allowed to walk passive movements of the thigh should be made, and these should gradually be increased in amplitude. About a week after the patient has put his feet to the ground he should be allowed to walk, and it is not advisable to keep the patient in the erect position unless he is walking, as the muscular movements involved in walking favour the circulation of the blood in the veins. When the patient begins to get about during convalescence he should be instructed to keep his legs up as much as possible in the intervals, and not to sit with his legs crossed.

If there be any delay in convalescence massage may be tried. A crêpe bandage will usually be found to answer much better than the elastic bandage formerly so much in vogue. Some of the foreign spas, notably Bagnolles-de l'Orne, Bagnères-de-Bigorre, Brides-les-Bains, Plombières and Bourbonnes-les-Bains have a great reputation in the treatment of cases in which complete recovery is delayed; this is especially the case in gouty patients. In this country I would recommend Droitwich and Harrogate as most suitable.

Mr. Foulds, of Droitwich, has favoured me with the particulars of a case of bilateral thrombosis of femoral and iliac veins in a man, æt. 33 years, as a result of typhoid fever, who derived great benefit from a course of warm saline baths at Droitwich.

For those who are good sailors a sea voyage is the ideal means for picking up strength after an attack of thrombosis.

It will be noted that I have said nothing about the surgical treatment of thrombosis, for the very good reason that I have had no experience of it, and from what I have read about it I gather that the risk to the patient is greater than if purely medical treatment be carried out. The same applies to the treatment of the disease by the subcutaneous injection in the neighbourhood of the vein of various preparations of silver, gold, platinum and palladium. I am distinctly in favour of abstaining from any active treatment of the affected part.

I have been obliged to omit reference to the treatment of the complications of femoral thrombosis, such as pulmonary embolism and infarction, as I fear that I have already occupied more time than I should have done.

April 5th, 1909.

THE OPERATION FOR BREAST CANCER.

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IT is possible that in the future the treatment of cancer will be carried out by the agency of some selective poison, which, acting like quinine in malaria, will pick out and kill the carcinomatous epithelium without destroying the normal tissues. When that happy time arrives, the problem how cancer spreads will recede into insignificance. But for the present, and so long as surgery remains the only therapeutic resource of any value, this question of dissemination is of the utmost practical importance. If we attempt to eradicate a plant, with all its roots, we must first find out its mode of growth, and how deep or how wide its roots may be expected to extend. We ought to know whether the young plants which spring up around it arise from seeds propelled to a distance, or whether they are offshoots from the roots of the parent plant.

According to the embolic theory of dissemination, the secondary nodules which spring up around a primary carcinoma do not arise in continuity with it. They arise from cancerous particles swept along by the force of the stream in the blood and lymphatic-vessels until they lodge somewhere or other and produce a metastatic nodule.

A close study of the evidence shows that secondary nodules do not arise in this haphazard way. They appear first close to the growth, and they spread from it in a centrifugal fashion, occupying a circle of ever-widening area and primarily attacking one particular plane of tissue—the plane of the lymphatic plexus into which the cancerous organ drains.

Evidently the embolic theory fails altogether to account for these peculiarities of dissemination. The only true metastases in cancer, that is to say the only satellite nodules produced by the embolic transport of cancer particles, are the deposits found in the lymph-glands. All other deposits appear to be formed by the actual growth of lines of cancer-cells in centrifugal fashion along the lymphatic plexuses, with which the primary growth is in relation. To this process, which must be carefully distinguished from infiltration, I have given the

name of "permeation." To complete this brief account of dissemination, it must be stated that as soon as permeation reaches the subserous lymphatic plexuses, either of the pleura or of the peritoneum, cancer-cells escape into these cavities and soon implant themselves upon the adjoining viscera. The progress of dissemination, hitherto slow, now becomes headlong, and the patient dies with multiple deposits upon the surface of those viscera which are in relation to the invaded serous cavity.

This view of dissemination, which may be called the permeation theory, first started three years ago, has met with much acceptance, although the embolic theory still finds a few supporters. Prof. Halsted, who is the leading American authority on breast cancer, has lately given his adhesion to my views, a fact which enables me to attack his method of operation with increased confidence, though with a worse grace.

One of the essential points of the Halsted operation is the removal of a most extensive area of skin, an area so large that skin-grafting is necessary to cover the gap. As a matter of fact, pathology and surgical experience unite to show that in the ordinary case of breast cancer it is sufficient to remove a circle of skin four, or at the most five, inches in diameter. The surgeon who removes an extensive area of the healthy skin surrounding the infected area may congratulate himself on his vicarious heroism, but he has done an ill service to his patient.

In this connection it is interesting to study why Halsted advised such an extensive ablation of skin. Even after removing considerable areas of skin he found recurrent nodules springing up in the surrounding integument. He inferred very naturally, but quite erroneously, that the growth was spreading in the plane of the skin, a belief which has been general up to a very recent period, and one which still holds its ground to some extent. To support this belief an entirely imaginary lymphatic plexus known as the deep cutaneous lymphatic plexus of Arnold has been assumed to exist, in spite of the vehement and well-founded denials of Sappey, the great French anatomist.

In order to avert these local recurrences in the surrounding skin Halsted began to remove the skin very extensively, but he was unable to bring his percentage of skin-recurrence below 16 per cent.

In point of fact, though at first sight the statement appears a paradox, Professor Halsted's skin-recurrences are not due to an insufficient removal of skin, but to an insufficient removal of the tissues beneath the skin, in which lies the deep fascial lymphatic plexus, the main highway along which cancerous permeation spreads centrifugally from the breast. He carries his skin incision at once and everywhere through the fat, straight down to and through the deep fascia. It is here that Halsted's operation errs capitally by defect.

Professor Halsted has been cutting off the head of the thistle instead of going to its roots. He does not undermine his skin-incisions at all so as to fashion skin-flaps. The area of deep fascia which he removes is exactly of the same diameter as the area of skin which he ablates. Consequently, unless the case be an early one, he leaves entirely untouched the microscopic growing edge of the cancer which I have succeeded in demonstrating beyond the region of visible secondary nodules. This microscopic growing edge consists of a ring-shaped area of permeated lymphatics belonging to the deep fascial lymphatic plexus. The skin-nodules whose occurrence Professor Halsted has to deplore so frequently are efflorescences from the intact "microscopic growing edge" in the deep fascia, and are directly due in most cases to the defects of his method of operation. If permeated lymphatics are left in the deep fascia, cylinders of cancer-cells push their way up towards the skin here and there along the tributary lymphatics which run down vertically from the skin to the fascial plexus. Continuing to advance towards the skin in a direction opposed to the natural flow of the lymph-stream, the plug of cancer-cells reaches and penetrates into the bundle of fine cutaneous lymphatics, in which each of these cutaneous tributaries takes its origin. The result is a localised spherical nodule of growth—a subcutaneous nodule. To prevent the formation of such nodules the skin-flaps must be extensively undermined, and a large area of the infected deep fascia must be excised. Fortunately the remova of a circular area of the deep fascia ten to twelve inches in diameter can be carried out even in patients of advanced age, not only without imperilling the life of the patient, but without increasing the shock of the operation. Two conditions only must be strictly observed, the

avoidance of sepsis and the avoidance of chill. The field of operation, except that part of it where the operator is at the moment engaged, must be kept covered with relays of hot towels without regard to the convenience of spectators.

Recurrence in the internal organs after operation is not generally regarded as the result of defects in the operation, unless at the same time external recurrence takes place. In many cases such an assumption is unsound. In a considerable proportion of cases of epigastric invasion no clinically recognisable nodules can be felt over the abdominal wall even in the latest stage of the disease. Recurrence in the abdominal viscera after operation may be due to the failure of the operator to remove a microscopically invaded portion of the epigastric deep fascia, even though no subcutaneous nodules of growth subsequently appear in the epigastric region to indicate the incompleteness of the operation. The circle of deep fascia removed should accordingly always include a greater or less extent of the anterior layer of the rectus sheath.

If breast cancer spreads by centrifugal permeation with approximate equality in all directions, but with a tendency to keep in certain planes predetermined by the arrangement of the lymphatic vessels, there is one great principle to be observed in its operative treatment—a principle which has never hitherto been fully carried out. It is essential that the primary growth shall always be the central point of the mass of tissue which is cut out. Breast cancer spreads centrifugally in the plane of the deep fascia, the skin and deeper structures being usually infected secondarily and less extensively. Therefore the deep fascia should be removed more widely than the skin and deeper structures. Of course it is essential to remove all the glandular tissue of the breast with the embolically invaded lymphatic glands and the trunk lymphatics draining the area concerned. Recurrence after operation is due to failure to remove all the permeated deep fascia rather than to removal of insufficient skin.

I now propose to detail the steps of the operation for breast cancer which I have devised to fulfil the requirements of the permeation theory. And I would say at once that while for other varieties of cancer my view of dissemination remains a highly probable theory, for breast-cancer it is proved up to the hilt by the four years' work which I have devoted to the subject. That breast-cancer is mainly disseminated by centrifugal permeation of the lymphatics is a proved fact which no surgeon can afford to ignore.

The skin incision.—There are two objects: first, to isolate the infected area of skin with a view to its subsequent removal, and second, to give access to deeper structures. The first incision, surrounding the infected area, is a circle made without reference to subsequent suture lines. The other parts of the incision are linear and should be planned to give the best access to deeper structures and to prevent the subsequent scar from causing undue discomfort. A rectilinear scar running along the anterior border of the axilla tends to bind the arm to the side and causes great discomfort. With such an incision it is also more difficult to bring the skin edges into apposition, all the tension acting in one direction, at right angles to it. The skin incision should therefore avoid the anterior boundary of the axilla. The best incision, which is only just deep enough to open up the subcutaneous fat, may be said to consist of three parts: (1) A ring incision, four to five inches in diameter, accurately centred on the growth and slightly trailing off into (2) above and (3) below. (2) A curvilinear incision for giving access to the axilla. This begins at the lower edge of the great pectoral near its insertion, and ends—also at the lower edge of the great pectoral—by joining the annular incision (1). Crossing the base of the axilla it marks out an almost semi-circular flap of skin whose convexity reaches back nearly to the edge of the latissimus dorsi. A flap consisting only of skin and subcutaneous fat is turned up, the base of the flap corresponding to the anterior edge of the pectoralis major. (3) A linear incision coming off from the lower and inner part of the annular incision and passing downwards for about two inches along the linea alba. This incision gives access to the deep fascia of the epigastric region, removal of which is essential. The skin-flaps are then undermined in the mid-plane of the subcutaneous fat until a circle ten to twelve inches in diameter is exposed. In the centre of this circle is the primary growth. The retracted skin-flaps should be carefully wrapped in hot towels.

Delimitation of the area of deep fascia to be removed.—An annular incision marking out the ten-inch circle of deep fascia to be removed is now carried down to the muscles. The deep fascia is

dissected all round from the underlying muscles, working from the periphery to the centre of the field of operation. It is unnecessary to carry this dissection further than the margins of the muscles which require removal, *i. e.* the pectoral part of the pectoralis major and the lower part of the serratus magnus—the portion which touches the deep aspect of the mamma. When the fringe of deep fascia has been raised to this extent, a blunt instrument is thrust between the clavicular and the costal portions of the great pectoral. The costal origins of the muscle are raised by a finger thrust beneath them, and are successively divided close to the costal cartilages. The muscle is rapidly stripped from the chest, exposing the origin of the pectoralis minor, which is also divided. A finger is now thrust beneath the insertion of the great pectoral, along the outer wall of the axilla, and one blade of a pair of scissors is passed along the finger. The insertion of the costal great pectoral is divided by one cut of the scissors. The insertion of the lesser pectoral is next divided close to the coracoid process, the axillary vessels being previously protected by a finger thrust beneath the muscle. The whole mass of breast and muscles is allowed to fall outwards. The arm is now somewhat approximated to the side, and the remaining clavicular part of the great pectoral is dragged upwards by a retractor or divided transversely if necessary. The costo-coracoid membrane, thus fully exposed, is carefully divided just below the clavicle, and the vessels and nerves to the pectoral muscles are severed where they pierce it.

Removal of axillary contents.—Access is thus obtained to the very apex of the axilla, perhaps the most crucial point of the operation, since here are situated the subclavian glands. Failure to remove these glands is a very serious and not uncommon fault of technique in the operation. It is generally followed by recurrence just below the clavicle.

The axillary vein is now sought for at the apex of the axilla and carefully cleared from above downwards. The axilla is then systematically cleared of all fat and glands, care being taken to preserve the subscapular nerves and the nerve of Bell. The digitations of the serratus magnus, which are overlaid by the outermost part of the breast, are finally divided, first at their origin and next at a point near their insertion into the scapula. Some years ago my senior colleague, Mr. Henry Morris, pointed out the necessity of removing at least that portion of the serratus magnus which lies in direct contact with the breast. The danger of cancerous infection of this muscle has hitherto been very generally ignored.

The parts removed form a single biconvex mass of tissue with thin extensive edges. To its outer side are attached the axillary fat and glands. Bleeding points in the field of operation are carefully sought for and ligatured, and two small drainage-tubes are inserted, one at the epigastric angle and the other at the posterior margin of the axilla. These are removed after twenty-four hours. Finally the skin edges are brought together in the best way possible. Usually there is little difficulty in approximating them, the extensive undercutting having rendered the flaps very mobile.

Except to those who have seen the operation and have observed the patient's pulse immediately afterwards, when it is almost invariably of good volume and below 100, often not more that 80, it may seem incredible that the operation described can be carried out with so small a degree of shock. The reason is a very simple one. The wide removal of the deep fascia so mobilises and frees the surrounding skin that the edges of the incision can usually be brought together, entirely without tension, by a single continuous suture of fine catgut or silk. This absence of tension ensures an efficient supply of blood to the flaps and a painless and rapid convalescence. Formerly the operator considered himself fortunate if he could drag the skin edges together by main force, and maintain them in position by thick sutures strained almost to breaking point. An Indian fakir impaled upon and hanging from an iron hook might almost be envied by contrast with a patient in such a condition, though his feelings would be very similar. My results have proved that the persistent shock, acute pain, and prolonged rapidity and weakness of pulse which used to follow the operation for breast cancer, are due almost entirely to tension in the skin-flaps. After my operation the pulse falls to normal on the second or the third day, and there is no pain after the first twenty-four hours. The mortality of the operation when properly carried out does not exceed 2 per cent.

April 5th, 1909.

HOW TO PRESERVE THE PUPIL WHEN OPERATING FOR CATARACT.*

By C. BELL TAYLOR, F.R.C.S., M.D.Edin.

Mr. President and Gentlemen,—Here is a patient, æt. 82 years, who, six weeks ago, was blind from cataract. I operated at once upon his left eye the first day I saw him, and now he has excellent vision, can pick up a pin from the floor without glasses, and you cannot tell, even on the closest inspection, that the eye has been operated on at all, "*Summa ars est celare artem.*" I operated upon two other cases the same day—one a sad cripple, who had undergone amputation, the other a friend of mine in his eighty-third year, from Mansfield. This patient, as you see, presents no trace of operative interference, and if you ask him if he had any pain during the operation, or after, he will tell you "none whatever." These aged persons as a rule do much better when operated on than one would *à priori* suppose possible. Quite recently, for instance, I operated upon an important personage in his ninetieth year; he had been quite blind for eight years, and as he had been treated by experts in London the case was considered hopeless. None the less he recovered most excellent sight, both for reading and distance, within three weeks of operation on his right eye. Here is a specimen of his handwriting, as perfect as perfect can be. I have here a photograph of a patient who was exhibited in this room fourteen years ago suffering from blindness, the result of "*cataracta complicata accreta.*" Both eyes were operated on at the same time without iridectomy, and I learn to-day that although the case was considered absolutely hopeless by her medical attendants, including experts, she still retains the excellent vision which was at once restored by operation. A precisely similar case from the Worcester Blind Asylum was similarly treated with like results, the patient after fifteen years of asylum life, taking his discharge and assuming the *rôle* of an ordinary citizen. I have quite a long list of similar cases, and it is well known that I have, on various occasions, at the Royal College of Physicians, at the Clinical Society of London, at Moorfields, at the Ophthalmological Society, at the meetings of the British Medical Association, at this Society and elsewhere, presented to the notice of the profession, patients, the subjects of single or double extraction for cataract, in whom all trace of operative interference was conspicuously absent; the pupils in each case were central and movable, sight was excellent, spectacles were dispensed with by some of the patients, and it was not possible to say whether the extraction had been made upwards or downwards. Here is a patient, æt. 67 years, who twelve years ago was subjected to double extraction for cataract, since when, as a furnace man in the employ of the Midland Railway Company, he has been almost daily exposed to great heat and blinding glare, and yet so perfect is the result of the operation that it has been doubted, even by experts, whether he had been operated on at all. I once did a double extraction for a lady who subsequently consulted an eminent London specialist, who, not suspecting, failed to perceive that she had been operated on for cataract. The same thing once happened to myself in the case of a patient I had operated on years ago and who consulted me for another affection. "Have you been operated on for cataract?" said an eminent ophthalmic surgeon addressing one of my patients on exhibit at a meeting of the Ophthalmological Society. "Yes, I have, both eyes." "Well, where are your spectacles?" "I have got none." "But you can't see without spectacles?" "Oh yes, I can, quite well!" In fact the central and movable pupil, as in aphakial animals, seems to compensate for the loss of the lens, and it is a fact that some patients, after extraction for cataract, are able to read without glasses. This of course is the *summum bonum*, the *ne plus ultra*, not only of ophthalmic surgery, but of all surgery.

Gentlemen, it has been well said that to restore sight to the blind has ever been considered one of the grandest of human achievements, but to restore sight to the blind without causing pain, without administering anæsthetics, without shedding a drop of blood, and without leaving a trace of operative interference, must surely be grander still. Those who have had both eyes operated on prove that the result is not due to mere chance, or

* Delivered (with exhibition of patients) before the Nottingham Medico-Chirurgical Society.

fluke; they will tell you that they have had little or no pain during operation and none since. I can guarantee that no blood has been lost; and you can see for yourselves that there is no trace of operative interference. When operating I stand behind the patient, who is reclined, and I almost invariably use artificial light. I fix the eyeball with this little spear-shaped instrument, the "Pique de Pamard," which gives control with far less traumatism than that attendant upon the use of ordinary fixation forceps. I then enter the point of my trowel-shaped knife (which enables us to use the right hand for the left eye), in the cornea close to the sclerotic at the base of a flap, which, when completed, will comprise one half of the cornea. The knife is then pushed quickly across the anterior chamber until it emerges at a counter puncture similarly placed, sawed rapidly upwards, and then turned forwards so as to complete the section in the upper border of the cornea; the capsule is then freely lacerated through the natural pupil and the lens extruded by slight pressure at the lower border of the cornea.

It is a mistake to suppose that iridectomy prevents prolapse of the iris. Otto Becker's post-mortem researches have shown that when iridectomy is done there is always prolapse of the iris at the angles of the wound. In addition to other drawbacks, when iridectomy is done the lens capsule is apt to be trapped during the subsequent extraction, an accident which never happens when the iris is respected. If only a small portion of the sphincter of the iris is excised you can see the iris advance and recede, prolapse and fall back again during the subsequent extraction, just as if no iridectomy had been done; whereas if you do a large iridectomy you spoil the eye. It is not true that iridectomy permits of complete evacuation of cortical *débris*. You may secure a perfectly black pupil to-day, but to-morrow it will be suffused with dissolving cortex, which will often remain for weeks just as if no iridectomy had been done. It is a delusion to suppose that a preliminary iridectomy prevents subsequent glaucoma. The worst cases of glaucoma that I have seen in connection with extraction for cataract have occurred in eyes subjected to a preliminary iridectomy. A double operation of course increases the danger of infection, and some patients, having submitted to one operation without benefit to sight, are loth to submit to another. I once operated upon a celebrated master of hounds. I saw him for the first time at eleven o'clock at night and operated within the hour. He was all right within a brief space, and some weeks afterwards had resumed his strenuous life in the open. I have just done a double extraction for an aged patient from Boston, U.S., and last year I had quite a number of operations on patients from Utah and Salt Lake City.

Shall we ever be able to dispense with surgical operation in the treatment of cataract? "'Tis a consummation devoutly to be wished!" And no doubt much may be done in this direction. I will show you shortly a patient who has made a marvellous recovery under sub-conjunctival injections of iodide of potassium. It is a mistake to suppose that because a patient has cataract he or she must therefore be operated on at once. I have postponed operation myself in some cases for years with the happiest results. The late Charles Bader has recorded cases where patients, previously disabled through imperfect sight the result of cataract, have recovered sufficiently to be able to carry on their business for years under the influence of iodide of potassium and judicious dilatation of the pupil, and it is well known that Badall has treated quite a number of cases more or less successfully in like fashion.

April 5th, 1909.

Vaccine Treatment of Erysipelas.—The results of the use of a vaccine prepared from Fehleisen's *Streptococcus erysipelatos* are reported by G. W. Ross and W. J. Johnson. They employed the vaccine in fifty cases, and from this experience they firmly believe that when properly administered it exercises a specific and controlling influence on the course of the disease—preventing its spread, lessening its severity, and hastening recovery. In the first sixteen cases they followed the method of opsonic therapy, but the results were so satisfactory that in the remainder they felt justified in omitting the usual opsonic blood examinations and had equal success in the remaining cases, many of which were severe. They admit, however, that in certain severe cases the usual thorough blood examination may be required.—*Med. Record*, vol. lxxv, No. 12.

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